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535 NORTH DEARBORN STREET • CHICAGO 10, ILLINOIS

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NORCROSS B. M., *N. Y. State J. Med.* **51: 2356,** Oct. 15, 1951.

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An ideal design for many presbyopes!

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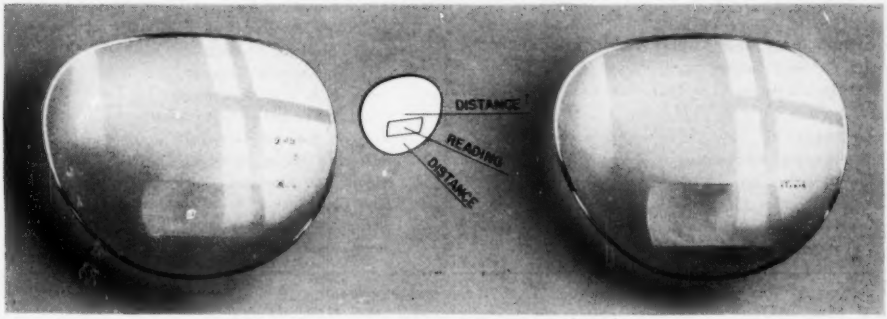

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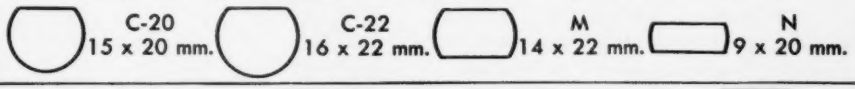
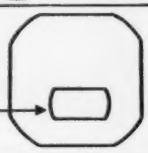
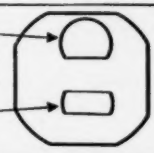


Regular
Select any reading seg design shown above.



Occupational
Any above seg design may be used in upper or lower portion as desired.



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Lectures:
Neuro-Ophthalmology
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Aqueous Secretion

Participants:
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Justin M. Donegan, Chicago
Joseph S. Haas, Chicago
Peter C. Kronfeld, Chicago
James F. Lebensohn, Chicago
Irving H. Leopold, Philadelphia
John M. McLean, New York
Samuel J. Meyer, Chicago
Maurice D. Pearlman, Chicago
C. Wilbur Rucker, Rochester
Derrick Vail, Chicago
Joseph A. C. Wadsworth, New York

Fee: $25.00 (Luncheons additional)
Registrar: Miss Maud Fairbairn, 8 West Oak Street, Chicago 10

The 8th Annual Gifford Memorial Lecture
8:30 P.M. February 23, 1952. The Drake Hotel
Dr. Alson E. Braley, Iowa City. Experimental Studies of Herpes Simplex Virus
Dinner 6 P.M., Open to all

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With its jewel-mounted plunger to minimize friction . . . inclined scale with mirror to overcome parallax error . . . new shape hammer . . . extra 7.5, 10 and new 15 gram weights . . . this truly excellent new instrument meets the specifications of the Committee on Standardization of Tonometers, American Academy of Ophthalmology and Otolaryngology . . . is individually tested and certified. In fitted case.

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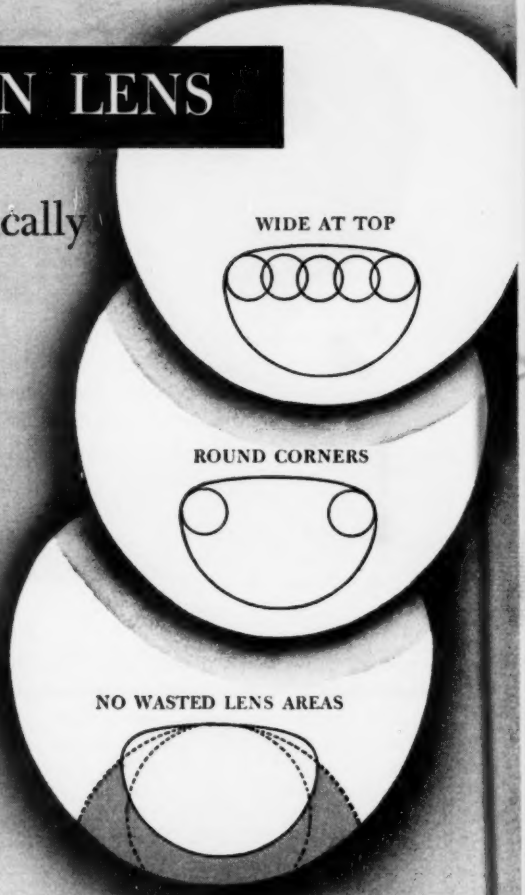

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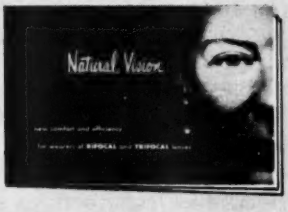

A new Bausch & Lomb booklet, "Natural Vision," tells what patients want in a bifocal lens, describes the characteristics which result in Panoptik's superior performance among bifocals, trifocals and special purpose multifocals. Ask your Bausch & Lomb distributor's representative.

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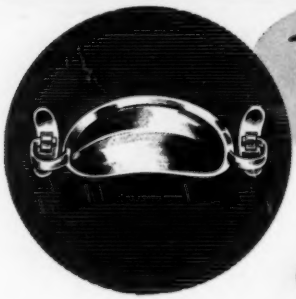

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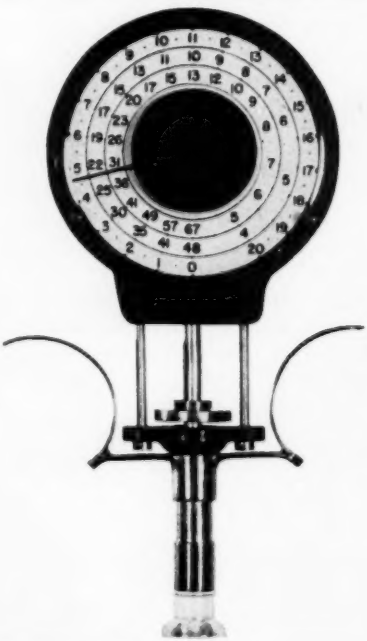

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1 to 4 p.m., January 14 through 18, 1952

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2 to 5 p.m., January 21 through 25, 1952

OPHTHALMOSCOPY
9 a.m. to 12 m., January 28 through February 1, 1952

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2 to 5 p.m., January 28 through February 1, 1952

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2 to 5 p.m., February 25 through 29, 1952

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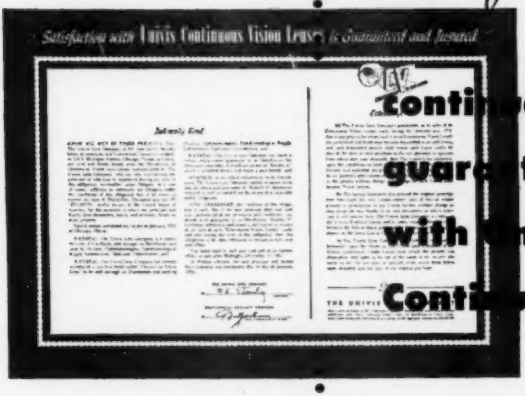

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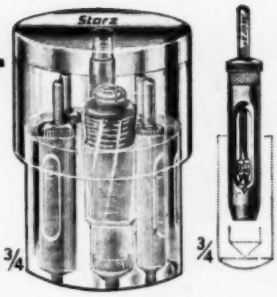

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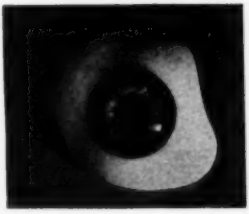

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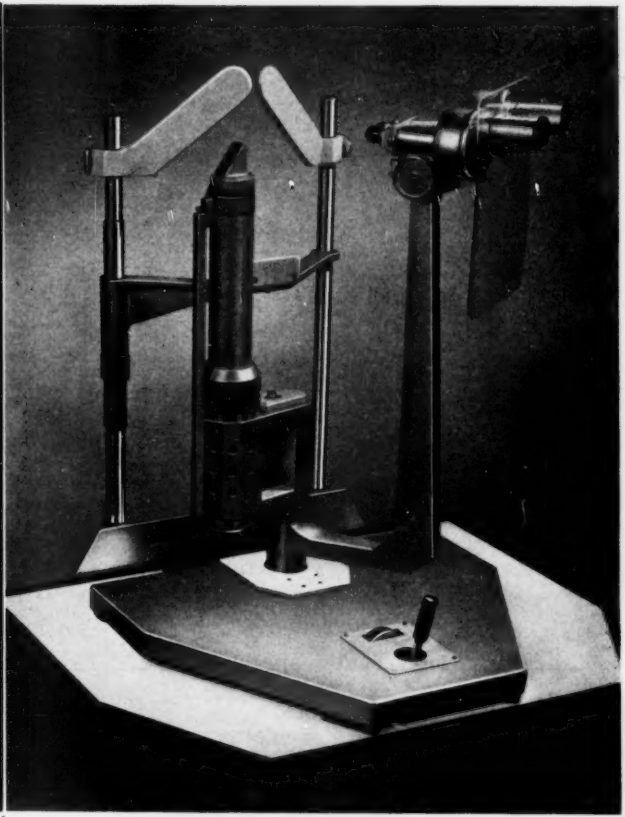

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NB: Owing to a printer's error, the range of observation and illumination was incorrectly given as 10°, instead of 180°, in our advertisement published in the November number.

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- Patients with dilated pupils can C/2 select frames.
- Provides Vision so presbyopes, hyperopes or myopes can C/2 select frames.
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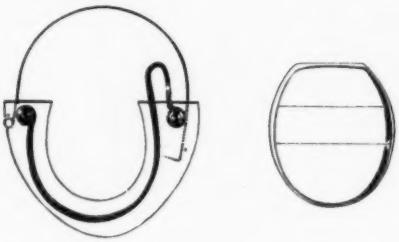

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Price $6.00 pr.

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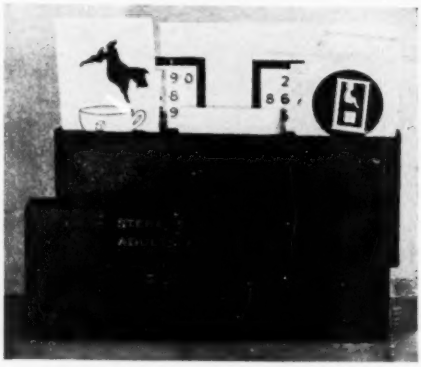

Can be used with the Stereocampimeter, Synoptoscope, Rotoscope and similar instruments; also with regular stereoscope. For adults and children.

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For Trial Frame

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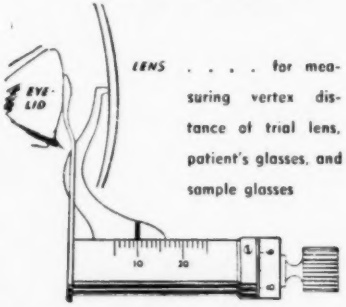

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ROLE OF HEREDITY IN ETIOLOGY AND TREATMENT OF STRABISMUS

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AND
BRUNO S. PRIESTLEY, M.D.
NEW YORK

ALTHOUGH it may seem a far-fetched endeavor to use the ponderous tool of genetics in the interpretation of everyday problems in strabismus, the hereditary approach, nevertheless, yields many clues in the etiology and management of strabismus. The aim of the present study was to analyze the multiple factors in the pathogenesis and etiology of strabismus and their transmissions. Genetics was used only as a tool for this purpose. Special attention was devoted to similar types of squint in the same family, with the hope that they may show similar patterns of response to surgical and orthoptic treatment.

REVIEW OF LITERATURE

Although it has been observed since the time of Hippocrates that, among other physiognomic traits, strabismus may be transmitted from parent to offspring, there have been relatively few studies analyzing the hereditary factors. Hippocrates [1] stated: "If then children with bald heads are born to parents with bald heads; and children with blue eyes to parents with blue eyes; and if children of parents having distorted eyes squint also for the most part . . ."

In more modern times, Böhm,[2] in 1845, was the first to emphasize the importance of heredity in strabismus. A quarter of a century later, Schmidt-Rimpler [3] and von Graefe [4] also called attention to its role. In 1901, Varese [5] described a mother and son with a form of retraction syndrome. Cohn [6] was the first to write a comprehensive paper on this subject. Of 805 patients with convergent squint, he found that 183 had relatives with strabismus.

From the Department of Motor Anomalies of the New York Eye and Ear Infirmary.

Read in part before the New York Academy of Medicine, Section of Ophthalmology, May 21, 1951.

1. Hippocrates: Airs, Waters and Places, in The Genuine Works of Hippocrates, translated by Francis Adams, New York, William Wood & Company, 1886, p. 171.

2. Böhm, L.: Das Schielen und der Sehnenschnitt in seiner Wirkungen auf Stellung und Sehkraft der Augen, Berlin, Dunker & Humblot, 1845.

3. Schmidt-Rimpler, H.: Augenheilkunde und Ophthalmoskopie, Ed. 7, Leipzig, S. Hirzel, 1901, p. 601.

4. von Graefe, A.: Motilitätstörung (1898), in von Graefe, A., and Saemisch, E. T.: Handbuch der Augenheilkunde, Ed. 2, Berlin, Julius Springer, 1939, Vol. 8, Pt. 1, p. 149.

5. Varese, P. M.: Vizio congenito ed ereditario della statica del bulbo oculare, Arch. ottal. **9**:143-146, 1901.

6. Cohn, H.: Über Vererbung und Behandlung des Einwärts-Schielen, Berl. klin. Wchnschr. **41**:1047-1051, 1904.

Parinaud,[7] Holthouse,[8] Lang and Barrett,[9] Worth,[10] and, more recently, Post [11] stressed the importance of inheritance in the etiology of strabismus. Parinaud stated that parents may transmit a disposition to strabismus without showing the trait themselves. According to him, antipathy to single vision is the most important of the hereditary factors and is frequently found in highly ametropic families. In his classic textbook on strabismus, Claude Worth emphasized that a defect in the fusion faculty is the essential cause of squint and is the inherited factor. "In the presence of this fundamental cause, the eyes are in a state of unstable equilibrium ready to squint either inwards or outwards on slight provocation." [10] Clausen and Bauer,[12] as well as Waardenburg,[13] concurred with Worth. Chavasse,[14] on the other hand, stated the belief that squint falls into two main types. The first type is characterized by hyperopia; the second begins as paretic strabismus and later becomes concomitant. In the former the error of refraction is the inherited factor; in the latter there is an inherited vulnerability of the embryonic neural epithelium.

Czellitzer,[15] who studied a large number of families, concluded that the mode of inheritance in strabismus is recessive. He believed that there are two inherited factors, and he attempted to work out, mathematically, the probabilities of inheritance for the different combinations of these factors. There are, however, in the literature family trees which show a dominant inheritance. These have been reported by Sicherer,[16] Landon,[17] Clausen and Bauer,[12] and Dufour.[18] Vogt [19] believed that the mode of inheritance is as an irregular dominance.

Anthropological studies shed further light on the inheritance and distribution of strabismus. Burnett,[20] Blitz,[21] Minor,[22] and Kollock [23] have commented on the rarity of strabismus among American Negroes. In a survey of specific diseases of

7. Parinaud, H.: Le strabisme et son traitement, Paris, O. Dion, 1899, pp. 56-60.

8. Holthouse, E.: Convergent Strabismus and Its Treatment, London, S. A. Churchill, 1897, pp. 13-15.

9. Lang, W., and Barrett, J. W.: Concomitant Convergent Strabismus, Roy. London Ophth. Hosp. Rep. **12**:133-148, 1889.

10. Worth, C. A.: Squint: Its Causes, Pathology, and Treatment, Ed. 5, London, Baillière, Tindall & Cox, 1923, pp. 54, 61.

11. Post, L. T.: Suggestions for the Surgical Management of Strabismus, Am. J. Ophth. **32**:345-350, 1949.

12. Clausen, W., and Bauer, J.: Beiträge und Gedanken zur Lehre von der Vererbung des Strabismus concomitans, Ztschr. Augenh. **50**:314-334, 1923.

13. Waardenburg, P. J.: Das menschliche Auge und seine Erbanlagen, The Hague, Martinus Nijhof, 1932, pp. 103-109.

14. Chavasse, F. B.: Worth's Squint, or Binocular Reflexes and the Treatment of Strabismus, Seventh Edition, edited by F. Bernard Chavasse, Philadelphia, P. Blakiston's Son & Co., Inc., 1939, pp. 308-310.

15. Czellitzer, A.: Wie vererbt sich Schielen? Arch. Rassen- u. Gesellsch. Biol. **14**:377-394, 1922-1923.

16. Sicherer, W.: Weiterer Beitrag zur Vererbung des Schielens, München. med. Wchnschr. **54**:1231, 1232, 1907; **56**:2707, 1909.

17. Landon, E. B.: Hereditary Transmission of Squint, Brit. M. J. **2**:1228, 1909.

18. Dufour, O.: Rev. méd. Suisse Rom. **29**:322, 1909.

19. Vogt, A.: Über Vererbung von Augenleiden, Schweiz. med. Wchnschr. **53**:188-193, 1923.

20. Burnett, S. M.: Am. J. Ophth., St. Louis **4**:299, 1887.

21. Blitz, Am. J. Ophth., St. Louis **4**:299, 1887.

22. Minor, James L.: Some Impressions of Certain Eye Affections in the Negro as Compared with the White Race, Tr. Am. Ophth. Soc. **12**:460-465, 1910.

23. Kollock, C. W., in discussion on Minor,[22] p. 465.

the eye among white persons and Negroes of the Federal Farm Security Administration borrower families,[24] there were approximately 2.50% of white persons with strabismus, but only 0.60% of Negroes. Holm[25] found 0.52% of persons with concomitant strabismus among 931 people studied in Gabon, Africa. In most of these the squint was of the alternating divergent type. Some authors have claimed that the low incidence of strabismus reported among Negroes is due to their rarely seeking medical attention for this condition.

PRESENTATION OF DATA

Of 158 patients with strabismus observed in the Department of Motor Anomalies of the New York Eye and Ear Infirmary, 75, or 47.5%, belonged to families with two or more members affected with squint. It is interesting to note that Worth[10] found 51.78% of patients with a family history, and Scobee,[26] 41%. These figures are probably smaller than the actual number because many patients with only a slight degree of heterotropia are missed.

There were 139 patients with esotropia, among whom 68, or 48.9%, had a family history of the disorder. This is in contrast to Cohn's finding[6] that 22.6% of patients with convergent strabismus had one or more relatives with squint. Among 19 patients with exotropia, we found 7, or 36.8%, with a family history of the disorder.

The present study consisted of 88 families (1 family with Duane's retraction syndrome). Eight families were from private practice[27] and were included in order to enrich our data with pertinent cases.

In 58 families we were able to examine two or more relatives with strabismus. In order to facilitate the presentation of this material, we have tabulated these data (Table 1). The patients have been grouped according to families; and the relationship, refractive errors, corrected visual acuity, type and amount of squint, and age of onset are shown. There were 101 cases of convergent strabismus, 3 of esophoria, and 2 of Duane's retraction syndrome. Of the cases of esotropia, 62 were of the monocular and 39 of the alternating type. There were 28 cases of divergent strabismus and 2 cases of exophoria. Of the cases of exotropia, 11 were of the monocular and 12 of the alternating type.

ETIOLOGIC CONSIDERATIONS

It is generally accepted that the etiologic factors in strabismus may be divided into three main types: anatomic, innervational, and fusional. From time to time various authors have placed greater emphasis on one or another of these elements. The 58 families tabulated in Table 1 were studied carefully to see which theories were supported by the facts.

The significance attributed to hyperopia in the causation of strabismus ranges from one of the greatest importance to a relatively minor role. Many authors following Donders emphasized hyperopia as the most significant factor in the causation of esotropia, while others, notably Worth, did not believe that hyperopia plays an important role.

While there is no doubt that the number of cases of hyperopia is far greater among patients with convergent strabismus than in the general population, it

24. Gover, M., and Yankey, J. B.: Physical Impairments of Members of Low-Income Farm Families—11,490 Persons in 2,477 Farm Security Administration Borrower Families, 1940, Pub. Health Rep. **59**:1163-1184 (Sept. 8) 1944; Reprint 2576.

25. Holm, S.: Le strabisme concomitant chez les palénégrides au Gabon, Afrique equatoriale française: Contribution à la question de race et de strabisme, Acta ophth. **17**:367-387, 1939.

26. Scobee, R. G.: Esotropia, Am. J. Ophth. **34**:817-833, 1951.

27. The following ophthalmologists furnished the pedigrees noted: Family 51, Dr. Adolph Posner; Family 47, Dr. Abraham J. Lanchner; Family 66, Dr. Hunter H. Romaine; Family 70, Dr. Arthur B. Duel; Family 80A, Dr. Bernard A. Roberts.

TABLE 1.—*Data on Fifty-Eight Families with Strabismus*

Family	Name and Relation*	Refraction	Vision	Type† and Amount of Squint (△)	Squint (△) sc‡	Squint (△) cc‡	Onset (Yr.)
1	Frank A. (B)	R: +1.00 ⌒ +1.25 × 80	20/30	R Et 50	..	..	..
		L: +1.00 ⌒ +0.50 × 90	20/30				
	Antin A. (B)	R: +4.00	20/30	Alt Et	60	50	4
		L: +4.00	20/30				
3	Elizabeth B.	R: −0.50 ⌒ −4.00 × 180	.....				
		L: +3.00 ⌒ +0.50 × 90	.....	R Et 75	..	..	Birth
	Stephen B. (F)	R: −1.25 ⌒ −0.75 × 160	20/20				
		L: +0.25 ⌒ +0.25 × 135	20/30	Alt Et 20	..	..	..
6	Bernice B. (S)	R: +2.50 ⌒ +2.00 × 90	20/30				
		L: +3.00 ⌒ +1.75 × 90	20/30	R Et	35	15	1½
	Joel B. (B)	R: +0.50 ⌒ +0.50 × 75	20/30				
		L: +0.50 ⌒ +0.50 × 105	20/30	Alt Et 20	..	..	4½
	Ruth S. (A)	R: +0.50	20/20				
		L: +0.50 ⌒ +0.25 × 90	20/20	Alt X-Xt 4-30	..	..	..
	Esther S. (A)	R	20/20				
		L	20/20+	Alt X-Xt 15-35	..	..	..
7	Janice B. (IT)	R: +2.00	20/30				
		L: +2.00	20/30	L Et 30	..	..	3½
	Jane B. (IT)	R: +1.00 ⌒ +1.00 × 90	20/30				
		L: +1.00 ⌒ +0.50 × 90	20/30	L Et 35	..	..	2
8	John B.	R: −3.25	20/30				
		L: −4.50	20/30	X 15	..	..	..
	Bennie B. (F)	R: −2.25 ⌒ −2.75 × 100	20/20				
		L: −1.25 ⌒ −1.75 × 90	20/30	Alt Xt 55	..	..	..
11	Philip B.	R: +1.75	20/30				
		L: +1.75	20/30	Alt X-Xt 25	..	..	..
	Barbara B. (M)	.....	.....	Alt Xt 30	..	..	..
	James B. (F)	.....	.....	Alt X-Xt 45	..	..	..
13	Alice C. (S) •	R: +4.50 ⌒ +1.50 × 90	.....				
		L: +4.00 ⌒ +2.50 × 90	.....	L Et 50	..	..	Birth
	James C. (B)	R: +4.50 ⌒ +0.50 × 90	20/20				
		L: +4.50 ⌒ +0.50 × 90	20/20	Alt Et 85	..	..	4
14	Robert C. (B)	R: +2.50	.....				
		L: +3.50	.....	L Et	45	10	3
	Elaine C. (S)	R: +4.50 ⌒ +1.00 × 90	20/40				
		L: +4.25 ⌒ +0.75 × 45	20/100	R Et 45	..	..	2
15	Joseph C. (B)	R: +1.50 ⌒ +1.00 × 180	20/30				
		L: +3.00	20/30	Alt Et 65	..	..	3
	Margaret C. (S)	R: +2.00 ⌒ +1.50 × 90	20/30				
		L: +2.00 × 90	20/30	L Et	..	..	..
17	Thomas C. (C)	R: +1.00 ⌒ +1.00 × 90	20/30				
		L: +1.00 ⌒ +1.00 × 90	20/20	Alt Et	38	17	4
	Robert F. (C)	R: +6.25 ⌒ +0.75 × 90	20/40				
		L: +3.00 ⌒ +0.50 × 90	20/20—3	R Et	24	10	7
18	John D. (B)	R: +0.50	20/40				
		L: +0.50	20/40	Alt Xt 25-50	..	..	..
	Jane D. (S)	R: +0.50 ⌒ +2.00 × 90	20/70				
		L: +1.50 × 90	20/70	Alt X-Xt 10-50	..	..	..
20	Jerome E. (B)	R: +2.00 ⌒ +3.00 × 115	20/70				
		L: +2.00 ⌒ +2.00 × 100	20/70	Alt Et 80	..	..	8 mo.
	Mary E. (S)	R: +1.50 ⌒ +0.50 × 180	20/200				
		L: +1.50 ⌒ +0.50 × 100	20/30	R Et 40	..	..	2½
21	James E. (B)	R: +5.00 ⌒ +2.00 × 90	20/70				
		L: +4.00 ⌒ +2.50 × 90	20/40	Alt Et 26	..	..	3 mo.
	Catherine E. (S)	R: +4.25 ⌒ +1.00 × 90	20/20				
		L: +5.00 ⌒ +1.00 × 90	Counting fingers	L Et 50	..	..	6½
	Lorraine H. (C)	Amblyopia, L. E.	.....	L Et	..	..	..
22	Donald F. (C)	R: +3.00 ⌒ +0.50 × 90	.....	L Et-E (acc)	15	E3	2½
		L: +4.50	.....				
	Karl K. (C)	R: +3.50	.....				
		L: +3.50	.....	Alt Et 50	..	..	Birth
23	Juliette F. (S)	R: +2.75 ⌒ +1.00 × 110	.....				
		L: +2.25 ⌒ +0.75 × 60	.....	E 10	..	..	..
	Seymour F. (B)	R: −3.50	.....				
		L: −3.50	.....	R Xt	..	..	Birth
23A	Julius F.	R: +0.50 ⌒ −1.00 × 180	20/20				
		L: +0.50 ⌒ −0.50 × 180	20/20	L Xt 25	..	..	6 mo.
	Samuel F. (F)	R: +0.50 ⌒ +0.50 × 90	20/20				
		L: +1.25 ⌒ +0.50 × 90	20/50	L Xt 40	..	..	..

TABLE 1.—*Data on Fifty-Eight Families with Strabismus—Continued*

Family	Name and Relation*	Refraction	Vision	Types† and Amount of Squint (△)	Squint (△) sc‡	Squint (△) ec‡	Onset (Yr.)
24	Gloria F.	R:+2.75 L:+2.75	20/70 20/200	Alt Et 35	..	..	2
	Lilian F. (M)	....	20/70 20/15	R Xt 20 Was Et	..	..	.. 2
25	Vito G. (C)	R:+1.25 × 90 L:+1.50 × 90	20/20 20/20	Alt Et 70	..	..	3 mo.
	Nancy T. (C)	R:+4.75 ⌒ +0.50 × 90 L:+5.00 ⌒ +0.50 × 90	20/30 20/30	Alt Et	30	15	3
26	Katie G. (S)	R:+3.75 L:+3.25	20/40 20/30	R Et (acc)	20	0	5
	Deborah G. (S)	R:+4.50 L:+4.50	....	Alt Et (acc)	14	0	5
29	Shiela G. (S)	R:+2.75 ⌒ +1.00 × 90 L:+2.00 ⌒ +1.00 × 90	....	Alt Et 38	..	..	2
	Cynthia G. (S)	R:+3.00 L:+4.00	....	L Et 38	..	..	3
30	Francine G.	R:−0.50 ⌒ +2.00 × 90 L:−1.25 ⌒ +0.50 × 90	20/40 20/30	R X-Xt 30	..	..	3
	Mable G. (M)	.....	.....	R Xt	..	..	..
33	Paulette H. (S)	R:+2.00 L:+1.50 ⌒ +0.75 × 135	20/30+ 20/30	L Et	35	9	2
	Paul H. (B)	....	20/20 20/20	Alt Et-E 12 (acc)	..	..	5
	Donald H. (F)	R:+1.50 × 180 L	20/40 20/50	R Et E (acc)	..	..	3
	Betty H. (M)	R:+4.25 ⌒ +0.25 × 180 L:+3.75	....	R Et	Et	E	..
34	Marilyn H. (S)	R:+1.50 ⌒ +0.75 × 90 L:+2.00 ⌒ +1.00 × 90	20/40 20/70	L E or Et 6	..	..	..
	James H. (B)	R:+5.00 ⌒ −1.00 × 45 L:+1.75	3/200 20/30	R Et 45	..	..	1½
	Ellen H. (G)	R L	1/200 20/70	R Et 60	..	..	..
37	Richard I. (N)	R:+2.00 L:+1.00	30/30— 20/200	L Et 40	..	..	3
	Carol M. (2C)	R:+3.50 L:+2.50	20/40 20/20	Alt Et 55	..	..	2
	Catherine I. (A)	R:−1.25 ⌒ −1.75 × 135 L:+1.00 ⌒ +0.50 × 90	20/70 20/50	R Et 45	.. ..	.. ..	.. 7
39	Paul K.	R:+7.00 L:+6.00	3/200 20/30	R Et 16	.. ..	.. ..	.. 2
	Sarah K. (M)	R:+1.25 L:+2.25	20/20— cf. (2m)	L Et 20	..	..	..
40	Maurice K. (C)	R:+4.00 ⌒ −1.50 × 180 L:+3.25 ⌒ −1.50 × 180	....	R Et 20	.. ..	.. ..	.. 2
	Frank C. (C)	R L	20/20 20/25	Alt Et 30	..	..	..
	Beatrice C. (M)	R L	20/20 Amblyopia	R Et 35	..	..	Birth
41	Hyman K.	R:+5.00 ⌒ +0.75 × 12 L:+4.75 ⌒ +0.75 × 90	20/40 20/30	R Et 40	..	..	..
	Sarah K. (M)	R:+2.00 L:+2.00	20/30 20/30	R Et 25	..	..	..
46	Anita L. (S)	R:+2.00 ⌒ +0.50 × 90 L:+1.50 ⌒ +0.50 × 90	20/20 20/20	Alt Et	25	10	Birth
	Michael L. (B)	R L	20/200 20/20	L Et 20	..	..	..
	Martin S. (C)	R:+3.00 ⌒ +0.25 × 75 L:+3.00 ⌒ +0.50 × 75	20/20 20/30	Alt Et	45	20	4-6 mo.
47	Judah L.	R:+1.50 × 90 L:+0.50 ⌒ −0.50 × 90	20/40 20/30	Alt Xt 28	..	..	4
	Martha L. (M)	R:−1.25 ⌒ −0.75 × 30 L:−1.25 ⌒ −1.00 × 165	20/20 20/15	L Xt 60	..	..	..
49	Rhoda L. (S)	R:+1.50 ⌒ −0.25 × 180 L:+1.50	20/20 20/40	Alt Xt 50	..	..	Birth
	William L. (B)	R:+0.75 L:+0.75	20/20 20/20	Alt X-Xt 15	..	..	Birth
	Mary L. (M)	R:−1.50 L:−3.00	....	X 10-30	..	..	..
	Esther S. (A)	R L	20/400 20/20	R Xt 40	..	..	..

TABLE 1.—*Data on Fifty-Eight Families with Strabismus—Continued*

Family	Name and Relation*	Refraction	Vision	Type† and Amount of Squint (△)	Squint (△) sc‡	Squint (△) cc‡	Onset (Yr.)
50	Arthur L.	R: +3.00 ⌒ +0.50 × 90 L: +4.00 ⌒ +0.50 × 90	20/30 20/30	Alt Et 80	..	..	..
	Alvin L. (F)	R L	cf. (2 ft.) 20/20	R Et	..	..	2½
51	Nathan L. (F)	R: −2.50 ⌒ −1.50 × 30 L	20/20 cf. (1 ft.)	L Xt 50	..	..	..
	Stephen L.	R L	20/25 20/20	Alt X-Xt 20	..	..	1½
53	Michael M.	R: +2.50 L: +2.00	20/50 20/70	L Et 55	..	..	3 mo.
	Margaret M.	R: −1.75 L: −0.50	cf. (10 ft.) 20/20	R Xt	.. 30	.. 15	.. ..
54	Lucille M. (IT)	R: −2.50 ⌒ −0.25 × 90 L: Plano	20/30 20/20	Alt Et 70	..	..	Birth
	Lorraine M. (IT)	R: Plano L: −1.75	20/20 20/20	Alt Et 70	..	..	Birth
55	Nicholas M. (B)	R: +3.50 L: +3.50 ⌒ +0.75 × 90	....	L Et 30	..	..	1
	Catherine M. (S)	R: +3.00 ⌒ +1.00 × 120 L: +2.50 ⌒ +0.50 × 180	20/40 20/20	R Et	.. 35	.. 8	.. 1
59	John M. (B)	R: +3.00 L: +3.00	20/100 20/40	R Et 8	..	..	..
	Mary M. (S)	R: +3.25 ⌒ +0.50 × 90 L: +3.25 ⌒ +0.50 × 90	20/200 20/50	R Et 35	..	..	..
60	Joan M. (S)	R: +3.00 L: +2.00 × 90	....	L Et 38	..	..	..
	Thomas M. (B)	R: +2.50 L: +3.50	20/30—2 20/200	L Et 65	..	..	1
61	William M.	R: +2.00 L: +2.00	....	R Duane Et 30	.. ..	.. ..	Birth ..
	Agnes M. (M)	R L	20/15 20/15	R Duane Et	..	..	..
62	Dion M.	R: +1.00 L: +1.00	20/40 20/70	L Et 80	..	..	Birth
	Janet M. (M)	R L	20/20 Amblyopia	L Et → Xt after surgery	..	..	..
63	Patricia M. (S)	R: +5.50 L: +4.75	20/40 20/70	R Et	45	10	3
	Michele M. (S)	R: +3.50 L: +3.00	20/30 20/200	L Et	35	22	3½
64	Arlene M. (S)	R L	20/40 20/40	Alt Xt 14	..	..	..
	Ellen M. (S)	R: +0.50 ⌒ +0.25 × 90 L: +0.50 ⌒ +0.25 × 90	20/30 20/20—1	Alt X-Xt	..	..	..
65	Mary M. (S)	R: +0.50 L: +0.50	20/30 20/50	LH 4	..	..	..
	Kathie M. (S)	R: +1.00 L: +1.00	20/100 20/30	R Et 35	..	..	6 mo.
	James M. (B)	R L	....	L Et	..	..	..
66	Thomas O. (B)	R: +3.25 ⌒ +0.75 × 105 L: +3.50 ⌒ +2.00 × 75	20/20 10/400	L Et	..	..	4
	Patricia O. (S)	R: +2.00 ⌒ +2.00 × 105 L: +1.50 ⌒ +1.50 × 95	20/25 20/20	Alt Et 35	..	..	6 mo.
	John O. (B)	R: +1.75 L: +1.25	20/30 20/15	R Et-E (acc)	Et 17	E 8	.. ..
	Margaret O. (S)	R: +1.00 × 90 L: +0.25 ⌒ +1.00 × 105 4 yr. later R: −3.00 ⌒ −0.75 × 180 L: +0.25 ⌒ +1.00 × 105	20/20 20/20 20/20 20/20	Alt Et 20	..	..	..
	Patrick O. (F)	R: −1.50 ⌒ −2.25 × 45 L: −2.00 ⌒ +3.75 × 75	20/40 20/60	E 6; was Et	..	..	..
67	Donald O.	R: +1.50 L: +2.75	20/20 20/200	L Et 85	..	..	1 ..
	Margaret O. (M)	R: +5.75 ⌒ +1.50 × 180 L: +6.25 ⌒ +1.00 × 165	20/30 2/300	L Et 70	..	..	..
68	Marie P. (C)	R: +3.50 L: +3.50	20/50 20/50	Alt Et	30	2	2
	Frank C. (C)	R: +1.00 ⌒ +0.50 × 90 L: +0.50 ⌒ +0.50 × 90	....	Alt Et 39	..	..	3 mo.

TABLE 1.—*Data on Fifty-Eight Families with Strabismus—Continued*

Family	Name and Relation*	Refraction	Vision	Types† and Amount of Squint (△)	Squint (△) sc‡	Squint (△) cc‡	Onset (Yr.)
69	Patricia P. (S)	R: +1.25 ⌒ +0.25 × 90	20/30				
		L: +2.25 ⌒ +0.50 × 90	20/40	L Et-E	20	12	..
	Theresa P. (S)	R: +5.00 ⌒ +0.50 × 90	20/25				
		L: +7.75 ⌒ +1.00 × 90	20/200	L Et	..	..	..
70	Joan Q. (IT)	R: +3.75	20/30				
		L: +3.75	20/100	L Et 20	..	..	1
	Jane Q. (IT)	R: +3.75	20/30	Alt Et 30	..	..	1
		L: +3.75	20/20	(LHt 15)			
71	John R. (B) v	R: +5.00	20/30		45	25	1
		L: +4.00	10/200	L Et	..	..	..
	Carmella R. (S)	R: +3.00	20/20	Alt Et 30	..	..	1
		L: +3.00	20/20				
	Rosemary R. (S)	R: +4.50	4/200	R Et	60	30	2
		L: +3.00 ⌒ +1.00 × 90	20/20				
	Joseph R. (B)	R: +3.00	20/50	R Et	50	30	1
		L: +3.00	20/50				
	Julia R. (S)	R: +2.75 ⌒ +0.75 × 30	20/40	Alt Et-E	Et	E	1
		L: +2.50 ⌒ +1.50 × 145	20/40	(acc)	20	9	..
	Rose R. (M)	R: +3.50 ⌒ +1.00 × 180	20/20				
		L	2/60	L Et 18	..	..	..
72	Carol R.	R: +3.50	20/30	Alt Et 20	..	..	Birth
		L: +4.00	20/30				
	Margaret R. (M)	R	20/20	Occ. Xt;	..	..	..
		L	cf. (3 ft.)	was Et			
74	Joan S.	R: +4.00	20/70+1	Alt Et 50	..	..	2½
		L: +3.75	20/30				
	Sylvester S. (F)	R: +2.50 ⌒ +0.50 × 90	20/30	R Et to R Xt	..	..	..
		L: +3.00	20/15	after surgery			
78	Patricia S. (S)	R: +0.75 ⌒ +1.00 × 180	20/200	R Et 18	..	..	..
		L: +0.75	20/30				
	Nancy S. (S)	R: +1.00 ⌒ +0.50 × 90	.....	Alt Et 38	..	..	Birth
		L: +1.00	.....				
79	Philip S. (B)	R: +4.25	.....	Alt Et 85	..	..	6 mo.
		L: +4.25 ⌒ +0.50 × 150	.....				
	Cynthia S. (S)	R: +0.75	.....		..	..	Birth
		L: +0.75 ⌒ +1.50 × 90	.....	L Et 20	..	..	..
80	Iola T. (S)	R: +4.50 ⌒ +2.00 × 90	.....	L Et-E	17	E 4	..
		L: +4.50 ⌒ +0.50 × 90	.....	(acc)			
	Marilyn T. (S)	R: +3.75	20/20	E 6	..	..	..
		L: +4.25	20/40—1				
	Carol T. (S)	R	20/20	Alt Et-E	14	E 5	..
		L	20/20	(acc)			
	Charles T. (F)	R: +7.00 ⌒ +1.00 × 65	20/70	R X-Xt 30	..	..	..
		L: +6.00 ⌒ +1.00 × 120	20/40				
	Walter W. (U)	R: +2.00 ⌒ +2.25 × 80	20/20				
		L: +1.00 ⌒ +2.00 × 85	20/50	L Et 10	..	..	..
	Sister C. (GA)	R: +5.00 ⌒ +1.00 × 145	.....	L Et to Xt	..	..	..
		L	cf. (2 ft.)	after surgery			
80A	Joel T. (IT)	R	20/20	Ortho	..	..	..
		L	20/20				
	Jonathan T. (IT)	R: +0.25	20/15				
		L: +1.50	20/70	L Et 12	..	..	..
81	Rocco V. (B)	R: +6.50	20/40	Alt Et 40	..	..	1
		L: +5.00	20/40				
	Gerald V. (B)	R: +2.50 ⌒ +0.50 × 50	20/30	Alt Et 70	..	..	Birth
		L: +2.75	20/20—3				
	Agnes V. (M)	R	20/20				
		L	Amblyopia	L Et	..	..	..
83	Dominick V. (B)	R: +2.50	20/30	Alt Et 75	..	..	..
		L: +2.75	20/40				
	Brenda V. (S)	R: +4.50	20/20	Alt Et 50	..	..	4
		L: +4.50	20/40	(RHt 15)			
84	Sarah V. (IT)	R: −1.75 ⌒ −0.25 × 90	20/20	Alt X-Xt 18	..	..	..
		L: −2.75 ⌒ −0.25 × 90	20/20				
	Anna V. (IT)	R: −2.25 ⌒ −0.25 × 180	20/20	Alt X-Xt 18	..	..	..
		L: −0.75 ⌒ −0.25 × 10	20/20				
	Betty V. (M)	R: +0.75	20/20				
		L: +2.25	20/20	L X-Xt 20	..	..	..
86	James W.	R: +3.25	20/20	Alt Et	70	40	..
		L: +3.00	20/30				
	Dorothy W. (M)	R	20/20				
		L: +1.00 × 180	20/50	L Xt 40	..	..	..

* A indicates aunt; B, brother; C, cousin; 2C, second cousin; F, father; G, grandmother; GA, great-aunt; IT, identical twin; M, mother; N, nephew; S, sister; U, uncle.

† Et indicates esotropia; Xt, exotropia; E, esophoria; X, exophoria; alt, alternating; X-Xt, intermittent, and/or periodic exotropia; Et-E, accommodative esotropia corrected by glasses to esophoria, Duane, retraction syndrome; Ht, hypertropia.

‡ Sc means without correction; cc, with correction.

would be a grievous error to assume that there is a constant ratio between the degree of hyperopia and the amount of squint or the number of patients affected with strabismus. In extreme cases a patient with hyperopia of 1.00 D. has an esotropia of 70△, while another with hyperopia of 6.00 D. has an esotropia of 14△ (Table 2). In the present series, the highest mean average, of 51△, occurred in the patients with refractive errors of +2.25 to +3.00 D., while the lowest mean average was present in the patients with hyperopia of over 5.00 D. In both these groups there were cases of the accommodative type. The largest number of purely or prevalently accommodative squint occurred in patients with hyperopia of 3.25 to 5.00 D.

Since there is no direct relationship between the amount of hyperopia and the amount of strabismus or the number of cases, it is highly improbable that hyperopia alone is the cause of strabismus. Three other factors which may play a role are (1) hyperactivity of the center of convergence; (2) defective fusion faculty, and (3) gross anatomic anomalies.

TABLE 2.—*Correlation of Refractive Error and Amount of Esotropia*

Refractive Error (D.)	Amount of Esotropia (△)	Average Amount Esotropia (△)	No. of Cases	No. of Cases of Accommodative Squint
Plano to +1.00	10, 25, 30, 35, 35, 40, 60, 65, 70	41	9	..
+1.25 to +2.00	10, 18, 20, 20, 25, 25-10, 28, 35, 35, 35-9, 50, 50, 50, 70, 85	38	15	2
+2.25 to +3.00	16, 25, 30, 35, 40, 50, 60, 65, 65, 70, 70-36, 75, 80	51	13	1
+3.25 to +4.00	6, 15-0, 20, 20-0, 20-2, 23, 25-0, 30, 30-0, 30-10, 35, 35, 50, 60-20, 70-30, 85	36	16	8
+4.25 to +5.00	12-0, 15-0, 18-5, 25, 25, 25-0, 35, 40, 40, 40, 50-15, 56, 60, 80	45	14	5
Over +5.00	14, 16, 25-2, 40, 40, 45, 45, 45-0, 80-30	33	9	3
Total		40.9	76	19

Hyperactivity of the center of convergence could cause an excessive response to any amount of accommodational effort. The accommodation is not stimulated for distant vision in the presence of small amounts of hyperopia because it is not needed, nor is it put to use in cases of higher hyperopia because a maximal accommodational effort will not improve the vision. Furthermore, the effort cannot be sustained long enough to permit improvement in vision, and the patient often abandons completely the effort to accommodate. Consequently, the mechanism of accommodation has its greatest effect in patients with a refractive error between +2.25 and +5.00 D. (Table 2).

There were only two families in which all the affected members showed purely accommodative squint (Families 26 and 33). In patients with a moderate amount of hyperopia and purely or predominantly accommodative esotropia, one may assume that there is an inheritance of the refractive error rather than of the strabismus. While this may be true for Families 26 and 33, most of the patients with accommodative strabismus belonged to families in which other members had a type of strabismus in which the accommodative factor was less prominent or totally lacking.

From a hereditary point of view, if the fusion faculty were defective, one would expect to find, in the same family, as many persons with divergent as with convergent strabismus, especially when the refractive error is negligible. Our statistics

prove convincingly that there is a constant direction of deviation. In families with convergent strabismus, there may be members with alternating squint, members with monocular squint, members with accommodative esotropias, and members with esophoria, but all have convergent squint. The same holds true for divergent strabismus. There were only five exceptions to this general rule in our data. These will be analyzed below. Even in these five families there is no certainty that an inherited defective fusion faculty was responsible for the strabismus.

The constancy of the direction of deviation within the same family tree is immediately evident from perusal of Table 1. In Family 66 there were four children with strabismus. One had left esotropia with eccentric fixation; another, right accomodative esotropia with vision correctable to 20/30 in the poorer eye, and two, alternating esotropia. The father gave a history of esotropia in childhood, but at the time of the study, when he wore his glasses, his eyes were orthophoric. In Family 71 (Fig. 2) there were five children with esotropia. John and Rosemary had monocular esotropia with amblyopia of one eye. The mother had a similar type of esotropia. Joseph had right esotropia with equal vision in his eyes. Carmella had an alternating esotropia, while Julia had an alternating accommodative convergent strabismus, which became an esophoria when she wore her glasses. The wearing of spectacles corrected about half the strabismus in John, Rosemary, and Joseph.

Among the families with divergent strabismus, Family 49 demonstrates that a similar pattern may hold true for families with exotropia. One child showed an alternating exotropia. Her brother had intermittent, periodic, alternating exotropia. The mother had exophoria, while the aunt had right exotropia with amblyopia.

The five families in which both convergent and divergent strabismus were present in the same family tree must be carefully analyzed before they can be considered exceptions. At the time of examination of both the mother and the daughter in Families 24 and 72, the daughter showed esotropia, while the mother had divergent strabismus. In both these families the mother gave a history of convergent strabismus in childhood. These families are not included among the five exceptions.

In Family 53 the son had left esotropia, while the mother had right exotropia with deep amblyopia. Although we have no historical confirmation, it is possible that the right eye turned out only after it became highly amblyopic. A similar situation was present in Family 86, in which the son had alternating esotropia, while his mother showed left exotropia. However, her relatively amblyopic eye had a vision of 20/50.

In Family 23, the sister had compound hypermetropic astigmatism with esophoria, while her brother had right exotropia with myopia. In Family 80 (Fig. 2) four of five members had esotropia or esophoria. However, one member, the father of the three girls, with a refractive error of +7.00 sph. $\bigcirc$ +1.00 cyl., axis 65 in the right eye and +6.00 sph. $\bigcirc$ +1.00 cyl., axis 120 in the left eye had intermittent and periodic exotropia. In these three families (Families 86, 23, and 80) it is possible that there was inheritance of a defective fusion faculty, although in Family 80 it is quite probable that a man with exotropia married a woman who was a carrier for convergent strabismus. A great-aunt and an uncle on the mother's side had esotropia.

In Family 6 (Fig. 1) the brother and sister had esotropia. According to the history, the grandfather on the father's side had convergent squint. Two aunts on the mother's side had an intermittent and periodic exotropia. In this family it is

highly probable that there was an inheritance of convergent strabismus on the father's side and of divergent strabismus on the mother's side. In this case convergence would be dominant over divergence.

Although weakness of the fusion faculty is an integral part of strabismus in almost every case, it is rarely the sole etiologic factor in squint. In two families (Families 86 and 23) it was probably the main factor. In the other families with both convergent and divergent strabismus the inheritance could be interpreted in different ways with different degrees of certainty. It is significant that in families in which esotropia and exotropia coexisted it was always (with one exception, Family 23) the children who had the convergent strabismus, while the older generation had the divergent type. In two cases we had a definite statement on the part of the parents that the divergence developed later in life.

In only one family did an obvious anatomic anomaly play a significant role in the etiology of strabismus. In Family 61, both mother and son had Duane's syndrome, and other members of the family, who were not examined, apparently had the more ordinary types of convergent strabismus.

The basic question in the genetics of strabismus is to determine what factors are really inherited. We have analyzed the families in which strabismus may have been caused by purely accommodative factors, defective fusion faculty, or obvious anatomic malformations. We are now faced with the problem of interpreting the bulk of our data for which inheritance is proved without any doubt, but for which none of the aforementioned factors offers a satisfactory explanation. Any theoretical explanation of familial strabismus must be comprehensive enough to embrace the wide variety of strabismus occurring within the same family tree.

The higher centers, association tracts, nerves, and extraocular muscles are the tissues which determine the position of the eyes in the orbits. A simple measurement of the angle of strabismus will not reveal the true origin or nature of the strabismus in a particular case. It is of importance in heredity to know, as far as possible, which factors are responsible for the strabismus. If the nerve tissues are involved, it would mean that there is a hereditary defect of the ectoderm, whereas if the muscles, check ligaments, and fascial attachments are affected, it would point to a hereditary defect of the mesoderm.

There are several clues which point to a neurologic nature of strabismus, either coexistent in the same person or present in the same family tree. These are nystagmus,[28] mental retardation, stuttering, and left-handedness. Chavasse [14] gave a family tree in which bilateral congenital paresis of the external rectus was inherited in the father and son, while other members of the family showed strabismus of unknown types. It should be borne in mind that anatomic or physiologic blocks may exist at any level in the complex network of afferent and efferent nerve pathways between the eyes and the brain and may be hereditary.

The variations in size, thickness, and insertions of the muscles, check ligaments, etc., are hidden from observation and presurgical studies, but are revealed to the surgeon at the operating table. They are proof of the mesodermal origin of strabismus in a second group of cases. Some of these variations in the muscles may arise in the second month of embryonic life, when the paraxial mesoderm divides into the

28. In two family trees that are not reported here, there were cousins, one of whom had the usual type of esotropia, while the other had a horizontal nystagmus but no strabismus. In another family, a sister had a monocular esotropia; her brother had a horizontal nystagmus.

four recti and the two oblique muscles. For example, one of the medial recti may obtain an abnormally large share of the paraxial mesoderm, and the external rectus, an abnormally small share. In this case the medial rectus would be abnormally thick and the lateral rectus on the same side very thin.

If there is a muscular anomaly, it would seem, at first sight, that the strabismus would be present at birth, and the hypothesis outlined above would fail to explain the cases of squint which arise later in life. This is true if the muscular anomaly is very great, but when the variation is small the deviation will become apparent only after the neuromuscular reflexes and balances begin to be stabilized. In the formative stages it may take very little to upset the delicate balance and lead to the development of strabismus.

Anisometropia, Amblyopia, and Abnormal Retinal Correspondence.—Anisometropia has often been cited as a cause of strabismus. Yet, in the present study, there were only 7 patients with a significant amount of anisometropia among 120 whose refractive errors were measured. In Family 3 the father had a refractive error of —1.25 sph. ⌒ —0.75 cyl., axis 160 in the right eye and +0.25 sph. ⌒ +0.25 cyl., axis 135 in the left eye and an esotropia of 20△. He used his right eye for near and his left eye for distance. His daughter had an esotropia of 70△ with a refractive error of —0.50 sph. ⌒ —4.00 cyl., axis 180 for the right eye and +3.00 sph. ⌒ +0.50 cyl., axis 90 for the left eye. The daughter was less than 2 years of age at the time these measurements were taken. In this family anisometropia probably plays a definite part in the pathogenesis of the strabismus. In Family 54 (Fig. 2) the identical twins had a small amount of anisometropia. It is difficult to evaluate the role of this condition on the development of the squint in these twins.

In the other three families in which anisometropia was present in only one member, it is obvious that this refractive condition could not play an important etiologic role. Margaret O., one of the children in Family 66, showed a refractive error of +1.00 D., axis 90 for the right eye and +0.25 sph. ⌒ +1.00 cyl., axis 105 for the left eye when she was first examined, at the age of 9 years. At this time she already had alternating esotropia. Four years later the refractive error in the right eye changed to —3.00 sph. ⌒ —0.75 cyl., axis 180, while that in the left eye remained the same.

In Family 37 (Fig. 2), a niece had slight hyperopia, and her second cousin had more pronounced hyperopia, but the difference between the right and the left eye was 1.00 D. in each case. The aunt, however, had anisometropia, with 3.50 D. of difference between the eyes. Yet she did not have the greatest amount of strabismus.

In Family 34 (Fig. 2) the sister, Marilyn H., had no anisometropia, while her brother, with a refractive error of +5.00 sph. ⌒ —1.00 cyl., axis 45 for the right eye and +1.75 sph. for the left eye had an esotropia of 45 △. In this case the anisometropia was probably the deciding factor in changing the convergence tendency, inherent in the family, to a frank esotropia. The small number of cases with anisometropia in families with squint proves that this refractive condition is a relatively unimportant factor in the etiology of hereditary strabismus.

There is a controversy whether amblyopia is a cause or a result of strabismus. If amblyopia were the cause of strabismus, one would expect to find its presence in all members of the same family. On the other hand, if it were the result of strabismus, the presence or absence of amblyopia would depend upon the type and duration of the strabismus. In Table 1 there are many instances in which one

member of the family had equal vision in the two eyes, while another had amblyopia in one eye. A typical example is Family 72. The child had 20/30 vision in each eye with full correction. Her mother had 20/20 vision in the right eye and only finger-counting ability at 3 feet (9 cm.) in the left eye. When amblyopia is present in more than one member of the same family, it is often more pronounced in the older member. Our conclusion, therefore, closely parallels that drawn by Waardenburg,[13] i. e., that amblyopia is not inherited. For these reasons, we believe that amblyopia is the result, rather than the cause, of strabismus.

Abnormal retinal correspondence may be present in some members of the family; yet other members with strabismus may show normal retinal correspondence. This is in line with the generally accepted idea that abnormal retinal correspondence is developmental, rather than hereditary.

MODE OF INHERITANCE

Although the mode of inheritance has not attracted much attention, a review of the world literature shows that both dominant and recessive inheritance has been reported. Czellitzer,[15] who published the most extensive study on this subject, found recessive inheritance the rule. Landon,[17] Sicherer,[16] and Clausen and Bauer [12] reported families with dominant inheritance. Vogt [19] believed that strabismus is inherited as an irregular dominant.

The main difficulty in the study of human heredity is the relatively small size of the families and the possibility of the physician's observing directly only two, and occasionally three, generations. If one attempts to search further into the family history, he must depend on guesswork and hearsay evidence. For this reason, it is necessary that we pool the data obtained from many families in order to gain a better understanding of the mode of inheritance. The problem is further complicated by the fact that there are at least two genes which cause strabismus, the gene which affects the ectoderm and the gene which affects the mesoderm. It is probable that the two types follow somewhat different patterns of inheritance. Since it is impossible, at present, to differentiate between these types, the only practical approach is to begin the study with easily observable data and to consider all the patients with convergent strabismus in one category and all those with divergent strabismus in another.

For the present genetic study, we used only a random sample of consecutive admissions for strabismus to the Department of Motor Anomalies of the New York Eye and Ear Infirmary. There were 56 family trees with convergent strabismus, 14 family trees with divergent strabismus, 5 family trees with both convergent and divergent strabismus, and 1 family tree with the retraction syndrome.

According to the fundamental laws of heredity, there are essentially three modes of inheritance: dominant, recessive, and sex-linked. In the presence of recessive inheritance, if neither parent has strabismus and each is a carrier, the expected ratio would be three normal children for every child with strabismus. If *S* stands for the normal gene and *s* for the defective gene, the mating would be represented by the following formula $Ss \times Ss$. Thus, there would be three possible combinations among the children: 1 *SS*, 2 *Ss*, and 1 *ss*. Those children with *SS*, having two normal genes, would be normal and have normal heredity. Those with *Ss* (where *S* is dominant over *s*) appear normal but are carriers for strabismus. Only those children with *ss*, carrying two defective genes, will show strabismus. Thus, if we start with the assumption that we are dealing with a recessive trait, the ratio among the

families affected with squint should be three normal members to every member with strabismus. In order to keep the statistics unbiased, it is important to take a random sample from all the families in which strabismus is present, even those in which only one member is affected. For this reason, 62 families in which only one member had convergent strabismus and 14 families in which only one member had divergent strabismus were used in this study, in addition to the family trees in which two or more members had strabismus.

In Table 3 the number of patients actually affected with strabismus is compared with the number of patients who would be expected to be affected in accordance with the genetic laws of recessive inheritance (3:1 ratio). In studies of this nature we can never obtain the uniformity which is the prerogative of laboratory experiments. Since we are dealing with families of different sizes, another element of irregularity is added. To compensate for this irregularity, a much more elaborate statistical formula must be applied.

Genetic statisticians have devised various methods and formulas in order to cope with data of this type. We believed that the a priori method, as outlined by Kurt Stern [29] for $Dd \times Dd$ marriages, was the most suitable for our problem.

The expectation for D (normal) children is $p = \frac{3}{4}$ and that for d (affected) children is $q = \frac{1}{4}$. We wish to find the empirical expectation q' denoting the fraction of d children to be expected among the pool of sibships which actually come under observation. The total affected children make up the proportion q (i. e., $\frac{1}{4}$) of all sibships, detectable and undetectable. Since these affected children are concentrated in the detectable sibships, the proportion q' in these sibships is larger than q, namely, according to relation:

$$q = q' (1 - p^n)$$

Solving for the desired q':

$$q' = \frac{q}{1 - p^n}$$

Instead of the observable *fraction* q', it is useful to determine the expected average *number* of affected children per sibship. This number is obtained by multiplying q' by n, the total number of children in the sibship.

The mean value of this coefficient $q'.n$ varies with the size of the family. It has been calculated by using the formula mentioned above. It will be noted that the value $q'.n$ increases progressively from the smallest to the largest families.

Table 3 shows the application of this method to our data. The number of families (x) are tabulated according to their size; $q'n.x$ gives the total number affected which is to be expected. For example, there were 49 families with two sibs who had normal parents. To find the number of children with strabismus which one would expect by applying the 3:1 ratio, it is necessary to multiply 49 by $q'.n$, which in this case is 1.143. Thus, the number of cases with strabismus which would be expected in families with two sibs is 56.007. This type of calculation was carried out for all the families in which both parents were normal (Ss).

For convergent strabismus the number of cases expected is 198.903, while the actual number affected in this group was 175. It is possible that the reason for the smaller number of cases of strabismus observed is due to a lack of penetrance in some families. Some patients who have the genetic make-up of ss do not manifest strabismus. This is because unknown factors prevent the strabismus-carrying genes from exerting their influence on the further development of the defect, and the eyes

29. Stern, C.: The Principles of Human Genetics, San Francisco, W. H. Freeman & Company, 1950, pp. 133-135.

remain straight. This phenomenon, which can be detected statistically as a gap between the number of cases of squint expected and the number actually observed, is known as a lack of penetrance.

In Family 33 (Fig. 2), in which both father and mother have convergent strabismus, only two of three children have esotropia. According to Mendel's law, the combination would be *ss* × *ss*, and all the children should have strabismus. Although the normal child carries the genes *ss*, there is a lack of penetrance, so that these abnormal genes cannot effect the development of strabismus. A similar example is present in Family 80A. Although the identical twins have the same genetic make-up, only one has strabismus, while the other is normal.

With the same statistical method, the number of cases with divergent strabismus expected among sibs with normal parents was found to be 31.210. The actual number

TABLE 3.—*Comparison of Number of Patients with Strabismus Expected and Number Actually Affected*

Size of Family	No. of Such Families (x)	$q.^{x}n.$	Total Affected Expected ($q.^{x}n.x.$)	Total Affected Observed
A. Convergent Strabismus (Esotropia)				
1	20	1	20	20
2	49	1.143	56.007	54
3	35	1.297	45.395	42
4	17	1.463	24.871	23
5	13	1.640	21.920	18
6	4	1.825	7.300	4
7	2	2.020	4.040	2
8	2	2.222	4.444	3
9	2	2.433	4.866	4
10 or more	4	2.515	10.060	5
Total			198.903	175
B. Divergent Strabismus (Exotropia)				
1	3	1	3	3
2	12	1.143	13.716	12
3	6	1.297	7.782	7
5	3	1.640	4.400	6
8	1	2.222	2.222	1
Total			31.210	29

of cases of exotropia which were observed in our data was 29. It would appear that the penetrance is greater in divergent than in convergent strabismus, but the number of cases is too small to allow any definite conclusions. Family 11 showed a definite lack of penetrance. Although both father and mother had divergent strabismus, only one of the two sons had strabismus.

In order to give the reader a better idea of the mode of inheritance, we have selected 15 families with convergent strabismus, 3 families with divergent strabismus, and 2 families with both convergent and divergent strabismus and in Figures 1 and 2 have tabulated the inheritance of strabismus in these families.

Where convergent and divergent strabismus are coexistent in the same family, it is of great interest to know the outcome of this tug of war between the two antithetic tendencies. In Family 6 (Fig. 1) there were convergent strabismus on the father's side of the family and exotropia on the mother's side; yet both children had convergent strabismus. In Family 80 (Fig. 2) the mother's aunt and brother had convergent strabismus. The father, although he had a high degree of hyperopia, had

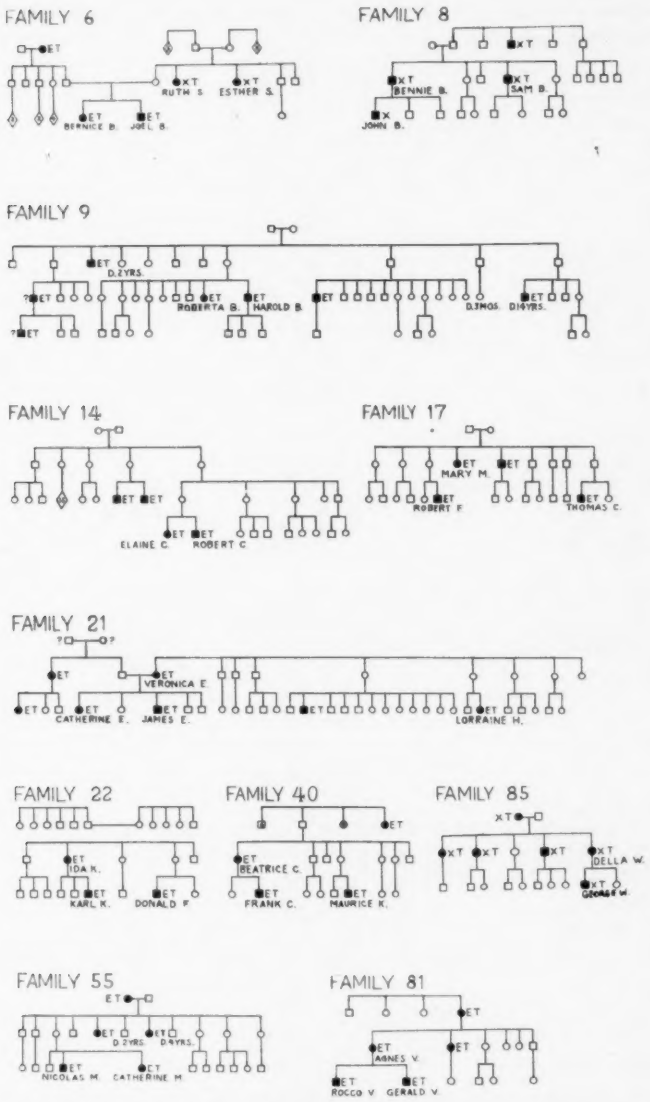

Fig. 1.—Pedigrees of Families 6, 8, 9, 14, 17, 21, 22, 40, 85, 55, and 81. *E*, indicates esophoria; *ET*, esotropia; *X*, exophoria; *XT*, exotropia.

intermittent and periodic exotropia. Two of these children had esotropia; the other had esophoria. From these two examples, it would seem that convergence is dominant over divergence.

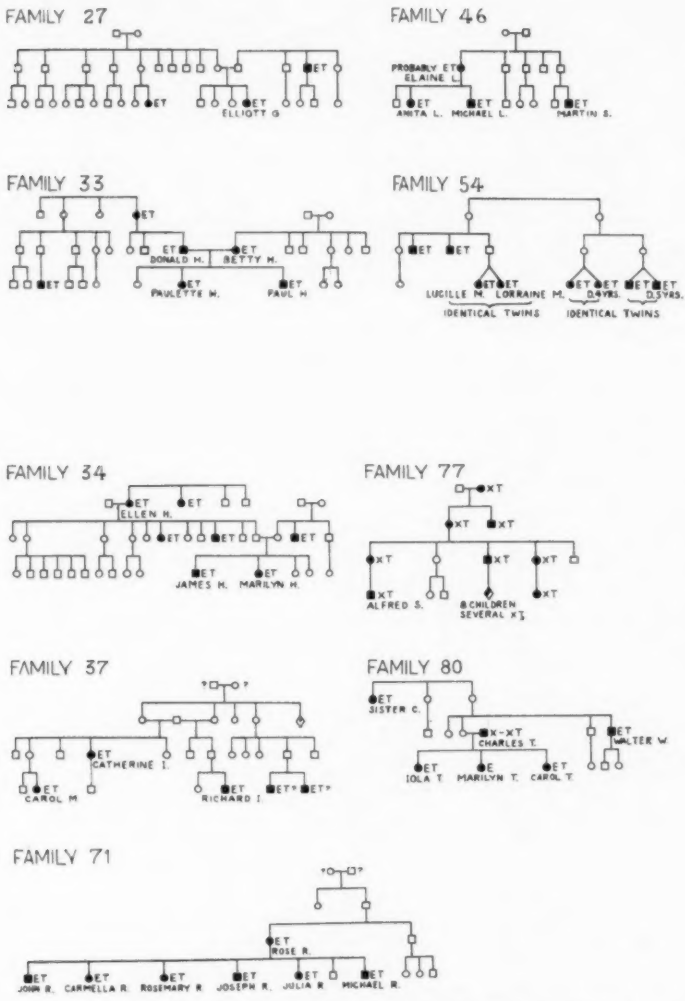

Fig. 2.—Pedigrees of Families 27. 46, 33, 54, 34, 77, 37, 80, and 71.

In order to determine the approximate number of carriers of strabismus, it is necessary to know the percentage of strabismus in the general population. The incidence of squint among 10,000 school children in Berlin was estimated by Cohn [6]

as 2%. Csapody-Mócsy [30] reported 4% with strabismus among 8,000 school children in Hungary. Barrie [31] analyzed the incidence of strabismus in British school children between the ages of 5 and 13. Of 22,890 children examined, 1.85% had strabismus. With an average figure of 2% for the proportion of persons with squint in the general population, according to the formula $2pq$,[32] the number of carriers (*Ss* persons) for strabismus is 1 out of every 4 persons, or 25% of the total population.

PRACTICAL APPLICATIONS

Since strabismus is inherited, it is not surprising that several members of the same family may show analogous types of strabismus and similar responses to treatment. The most obvious cases are illustrated by Families 26 and 33 (Fig. 2), in which all the members have accommodative strabismus which has been corrected entirely by glasses.

In the study of strabismus in a child, it would be of value in the management of his case to know what the future holds in store for him if he remained untreated. Very often it is necessary only to examine the parent or an older brother or sister (Families 8, 30, 47, 50, 51, 67, and 81). In Family 8 (Fig. 1) the son had exophoria of 15△. His vision was 20/30 in the right eye with a correction of —3.25 D. and 20/30 in the left eye with a correction of —4.50 D. The father had an alternating exotropia of 55△. Although he had never worn glasses, his refractive error was —2.25 sph. ⊂ —2.75 cyl., axis 100 for the right eye and —1.25 sph. ⊂ —1.75 cyl., axis 90 for the left eye. Vision was 20/20 in the right eye and 20/30 in the left eye with correction.

The son in Family 51 had periodic and intermittent exotropia. His vision was 20/25 with either eye, and he had no refractive error. The father had esotropia of 50△. Vision in the right eye was correctable to 20/20 with —2.50 sph. ⊂ —1.50 cyl., axis 30, while vision in the left eye was limited to finger-counting at 1 foot (30 cm.). In this case an overoptimistic ophthalmologist, who would expect the child to "outgrow" the muscle anomaly, might be cruelly disappointed when he discovered, after a few years, that the intermittently and periodically deviating eye had developed a permanent outward turn and deep amblyopia. Although there is no certainty that the same developmental pattern will be present in the father and the son, certainly much more caution and more frequent check-ups will be necessary in this patient, in whom the hereditary influence is present. Active intervention, rather than passive waiting, will become imperative as soon as it is established that the inconstant exotropia has become a permanent exotropia. On the other hand, in some families there may be a tendency for the strabismus to become less marked over the years. Four out of five members in Family 66 improved without surgical intervention over a period of 5 to 20 years.

The most important and controversial subject in strabismus is the correlation between the type and amount of squint and the type and amount of surgical correction. The results are often unpredictable, and a certain amount of skepticism

30. von Csapody-Mócsy, M.: Ein Beitrag zur Pathologie des Begleitschielens, Klin. Monatsbl. Augenh. **92**:385-387, 1934.

31. Barrie, T. S.: The Incidence of Squint at Various Ages, Tr. Ophth. Soc. U. Kingdom **42**:273-277, 1922.

32. Stern, C.: The Principles of Human Genetics, San Francisco, W. H. Freeman & Company, 1950, p. 160.

prevails among the most experienced muscle surgeons regarding the advisability of giving definite rules to guide the ophthalmologist in planning his procedure. There seems to be little relation between the measuring prism and the surgical caliper.

Table 4 shows the following correlations:

1. Equal amounts of surgical correction often give different results in different patients. One patient with alternating esotropia of 80△ (Family 20, Jerome E.) had correction with a bilateral medial-rectus recession of 3 mm. to esophoria of 10△ In another patient (Family 43, Kenneth L.) right esotropia, which measured 30△ for distance and 50△ for near, was altered, by a bilateral medial-

TABLE 4.—*Correlation of Type and Amount of Esotropia with Type and Amount of Surgical Correction in Ten Cases**

Name	Type	Esotropia Both Eyes (△)	Type and Amount Surgery (Mm.)	Results (△)
Family 9 Roberta B.	R Et	D:44 N:38	Rec RMR 2.5 Res RLR 7.5	D:Et 8 N:Et 8
Family 1 Frank A.	R Et	D:48; RH 3 N:52; RH 3	Rec RMR 3 Res RLR 8 Res LLR 8	D:E-Et 7 N:Et 7
Family 35 Geraldine H.	Alt Et	D:65 N:85	Rec RMR 3 Res RLR 8 Rec LMR 3 Res LLR 8	Xt 10 1 yr. later D:Et 25 N:Et 50
Family 52 Roberta L.	R Et	D:30 N:30	Rec RMR 3 Res RLR 8	D:Et 2-3 N:Et 4-6
Family 62 Dion M.	L Et	D:80 N:80	Rec LMR 4 Res LLR 8	D:Et 20 N:Et 20 D:Et 6 N:Et 6
Family 20 Jerome E.	Alt Et	D:Et 80 N:Et 80	Bil Rec 3	D:E 8-10 N:E 8-10
Family 21 James E.	R Et	D:Et 23 N:Et 30	Bil Rec 3	D:Et 2 N:E 1
Family 24 Gloria F.	Alt Et L Fix	D:Et 35 N:Et 35	Bil Rec 3	D:XT 35-40 N:XT 4-5
Family 43 Kenneth L.	R Et	D:Et 30 N:Et 50	Bil Rec 3 Bil Res R 9 L 5	D:Et 25 N:Et 25 D:Et 20 N:Et 25
Family 56 Susan M.	L Et	D:Et 30 c.c. 16 N:Et 30—20	Bil Rec 3	D:E-Et 2-3 N:E-Et 2-3

* Alt indicates alternating; Et, esotropia; E, esophoria; D, distance; Fix, fixating eye; N, near; Rec, recession; Res, resection; RMR, right medial rectus; RLR, right lateral rectus; LLR, left lateral rectus; LMR, left medial rectus; Bil, bilateral; E-Et, accommodative esophoria corrected by glasses to esophoria; XT, exotropia; c. c., with correction.

rectus recession of 3 mm., to 25△ of esotropia for distance and near. After a bilateral lateral-rectus resection the esotropia still measured 20△ for distance and 25△ for near.

2. Different types and amounts of surgical correction may give identical results in cases with similar measurements. Roberta L. (Family 52), with right esotropia of 30△, had correction to esotropia of 5△ by means of a 3-mm. recession of the right medial rectus and an 8-mm. resection of the right lateral rectus. James E. (Family 21; Fig. 1), with right esotropia of 26△, had correction by a bilateral medial-rectus recession of 3 mm. to an esotropia of 2△.

Therefore, any clue which enables one to foretell the end-results of surgical intervention gives a welcome addition to the data in a particular case. Consideration of the hereditary factors offers this type of aid. Although currently neglected,

the information one can glean from the end-results of surgery in one member of the family in which similar types of squint prevail is probably the safest guide in planning surgical correction on another member of the same family. Three examples illustrate this point.

In Family 40 (Fig. 1) the mother had right esotropia measuring 30Δ for distance and 40Δ for near. A 4-mm. recession was performed on the left medial rectus and an 8-mm. resection on the left lateral rectus. This can be considered a classic recession and resection, and most surgeons would agree that the amount was correct for the deviation measured. According to O'Brien's average figures,[33] in this type of case one would expect a correction of 32Δ. Unfortunately, however, in this patient the final result of the surgical correction was an exotropia of 40Δ. Her son, who had an alternating esotropia of 30Δ for distance and of 35Δ for near, had correction with a 3-mm. recession of the left medial rectus and an 8-mm. resection of the left lateral rectus. After the left eye had remained straight for a short period after the operation, it began to turn out, until he had an exotropia of 30Δ for distance and 50Δ for near. This overcorrection could not be rectified even by supplementary surgery which consisted of resection of the medial rectus and its reattachment to the original insertion. About 30Δ of exotropia still persists.

In Family 54 (Fig. 2), the surgical procedures on a set of identical twins proved to be an actual clinical experiment. Lucille and Lorraine M. were first observed at the New York Eye and Ear Infirmary in 1940, at the age of 4½ years. Both had alternating esotropia measuring more than 70Δ. A 5-mm. recession of the left medial rectus and an 8-mm. resection of the left lateral rectus were performed on Lucille. Ordinarily, one would expect a slight undercorrection from this amount of surgical intervention. Instead, a pronounced overcorrection resulted. The sister, Lorraine, who had an alternating esotropia with the same measurements and the same type and amount of surgical correction on the right eye, had the same marked final overcorrection. Unfortunately, the two girls were operated on the same day, so that the surgical response in the one could not be utilized in the treatment of the other.

The final example shows how the surgical result in the mother aided in the planning of operation on her son. In Family 62, the mother, aged 28, had had overcorrection operation in childhood for left esotropia. Her son, age 5½ years, had a left esotropia measuring 80Δ. The refractive error was negligible, measuring +1.00 in each eye. A 4-mm. recession of the left medial rectus resulted in esotropia of 20Δ. Two months later an 8-mm. resection was done on the left lateral rectus and resulted in residual esotropia of only 6Δ. Since 4 mm. of recession of the left medial rectus corrected 60Δ of strabismus, it is obvious that this correction is far greater than one would expect from the amount of the recession performed. Using the average figures given by O'Brien, that in high degrees of convergent squint 1 mm. of recession of the medial rectus corrects 7Δ of strabismus, one would expect the 4-mm. recession in this case to correct only 28Δ of strabismus. The gross overcorrection in the mother served as a warning to the

33. O'Brien, C. S.: Strabismus Ophthalmic Symposium, edited by James H. Allen, St. Louis, C. V. Mosby Company, 1951, pp. 388-393.

surgeon to proceed cautiously; as a matter of fact, notwithstanding the high degree of initial deviation, only one muscle was operated on in the primary procedure, and with good results.

SUMMARY AND CONCLUSIONS

Of 158 patients with strabismus, 47.5% belonged to families with two or more members affected with squint. The incidence was 48.9% among patients with esotropia and 36.8% among patients with exotropia.

Eighty-eight family trees were studied, and we were able to examine two or more members in 58 families.

Hyperopia alone was not the cause of convergent squint.

Hyperactivity of the center of convergence, defective fusion faculty, and obvious anatomic malformations, although important, were not major inherited factors except in a small percentage of the families.

The direction of squint always remained the same, with five exceptions. The latter was analyzed. The type of squint, however, might vary a great deal within the same family.

There are probably two types of inheritance: (*a*) a defect in the ectoderm, involving the nerve tissues, and (*b*) a defect in the mesoderm, involving such structures as muscles, check ligaments, and fascial attachments.

Anisometropia is a relatively unimportant factor in the etiology of familial strabismus.

Amblyopia and abnormal retinal corespondence are not inherited.

By use of the a priori method, it was found that the data fit a 3:1 ratio, indicating a recessive inheritance for both esotropia and exotropia. There was a certain amount of lack of penetrance in both the families with the convergent and the families with divergent strabismus.

Convergence is dominant over divergence.

About 1 out of every 4 persons is a carrier of the gene for strabismus.

By examining the patient's parent, it is sometimes possible to foretell what the future holds in store for the patient if he should remain untreated.

In the surgical management of strabismus, the behavior of the squint in one member of the family may serve as a fair guide to the response of another member to similar surgical correction, if the types of strabismus are the same in the two members.

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Miss Ann Brutton, of the Social Service Department, helped in contacting the different members of these families, and Miss Dorothy Parkhill and Miss Leta Counihan, orthoptic technicians at the New York Eye and Ear Infirmary, assisted in the study.

ABSTRACT OF PAPER
FURTHER EFFORTS TO INFLUENCE X-RAY CATARACT BY CHEMICAL AGENTS*

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NEW YORK

LAST YEAR my co-workers and I reported in a preliminary study on the relation of cysteine treatment to x-ray damage of the rabbit's eye which had been exposed locally to a single dose of 1,500 r of penetrating x-rays. There was at this time strongly suggestive evidence that among the signs of radiation damage to the eye two characteristic late effects of such radiation, epilation and x-ray cataract, were partially prevented by the intravenous use of 2 gm. of cysteine prior to irradiation.

These animals have been followed now over periods of 12 to 16 months. Total cataract has developed in the control group in 12 of 14 animals within the observation time of 14 months. None of the cysteine-pretreated eyes showed at this time a lenticular opacity which was dense enough or extensive enough to interfere substantially with the visibility of fundus details. However, all these lenses exhibited some fine ring- or star-shaped opacities in the posterior cortex and delicate lesions anteriorly which were not demonstrable photographically, but can be illustrated by drawings. These fine changes could not be remedied in intensity and character by local cysteine treatment prolonged for three weeks after irradiation.

In another series, 10 rabbits received as the only medication subconjunctival injections of cysteine in one eye, which preceded exposure of both eyes to the x-ray beam. This type of administration secured a concentration of the amino acid in the aqueous humor which was about one-fourth of the drug level obtained with intravenous injection. The protective effect of the locally applied cysteine in this

Read at the Eighty-Seventh Annual Meeting of the American Ophthalmological Society in White Sulphur Springs, Va., June 9, 1951.

*This paper was presented at the meeting of the American Ophthalmological Society in White Sulphur Springs, Va., and accordingly the manuscript cannot be published prior to the appearance of the *Transactions of the American Ophthalmological Society for 1951.* Studies on radiation cataract are being carried out in various laboratories by radiobiologists and radiophysicists, and it is believed that it would save considerable expense for the Government and other institutions carrying out such work if the various groups were kept informed of what has been done and presented at meetings throughout the country as soon as possible. In this way over-lapping and reduplication of experimental approaches can be avoided to some degree, or experimental approaches can be selected which might supplement completed studies for the best of the project. It is for this reason that the abstract of this paper is published.

—The Editors

group of experiments was definite but moderate in the majority of animals. The results are not without interest, especially in view of the high dose of x-rays which had been employed.

The second part of the study was concerned with a comparative analysis of microscopic signs of x-ray damage to the lens in cysteine-pretreated and control eyes. Thin meridional sections of lenses or flat preparations of the entire lens epithelium were obtained from more than 100 eyes and were used to follow initial changes at time periods ranging from two hours to three weeks after irradiation, before ophthalmoscopically visible changes had appeared. One of the earliest changes—the displacement of individual nuclei in the lens bow—occurred less readily in the cysteine-pretreated eyes, and a similar difference between the two groups of eyes was observed in cell counts in the preequatorial area. The incidence of x-ray-induced cell death in this peripheral zone was thus shown to be greater in the control eyes than in the protected eyes. Another early change—nuclear fragmentation with extrusion of Feulgen-positive material—was seen in flat preparations more frequently in the eyes which had not been pretreated with cysteine. Special efforts were made to study the effect of x-rays on the mitotic mechanism of the lens epithelium under the influence of cysteine treatment. It was determined that total flat mounts of the lens epithelium from the eyes of normal mature rabbits contained about 230 dividing cells and that the mitotic index of the lens epithelium is approximately 27 for 100,000 cells. Cysteine premedication altered to a moderate degree the temporary suppression of cell division, which lasts from two hours to three days after irradiation, the recovery from the mitotic inhibition, and the compensatory increase in the proportion of the dividing cells during the second week. It will be necessary to continue these laborious studies and to accumulate more data before many pending questions can be answered on this important topic.

It could be said, then, that all clinical and microscopic signs of early x-ray damage to the lens were influenced to some extent by the use of this chemical substance. This fact should be emphasized, since it indicates that indirect biochemical mechanisms of x-ray action, and not direct "hits," must be responsible, at least in part, for eliciting these lesions.

The last part of the study consisted of investigations in which chemicals other than cysteine were administered intravenously or locally. Four sulfur-containing and four sulfur-free compounds were used. The sulfur-containing compounds were glutathione, thiourea, sodium thioglycollate, and dimercaprol (BAL). The sulfur-free compounds were dihydroxyacetone, *dl*-alpha-tocopherol, citrovorum factor (folinic acid) and potassium cyanide. Only glutathione and thiourea exerted a beneficial effect on the x-rayed eyes. Most of the listed substances, or closely related compounds, have been reported as being effective in protecting bacteria or mice against the lethal action of x-rays. It can be concluded, therefore, that the results obtained on such material cannot be applied directly to the eye as far as the protection phenomenon on the lens is concerned.

MONOCULAR DIPLOPIA (BINOCULAR TRIPLOPIA) IN CONCOMITANT STRABISMUS

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THE PHENOMENON of monocular diplopia (or binocular triplopia) which is observed in some patients with concomitant strabismus consists essentially of the simultaneous localization of a single physical stimulus, reaching one eye, in two different subjective visual directions, one normal and one abnormal. As a consequence, patients who display this phenomenon have triplopia when observing with both eyes.

Binocular triplopia is rare as a spontaneous occurrence.[1] This is not surprising in view of the special conditions required for its appearance. Modern investigations with major amblyoscopes of patients with concomitant strabismus have shown, however, that one can relatively frequently elicit it artificially (Cass[2]). Indeed, Walraven[3] has developed a special orthoptic technique for the treatment of anomalous correspondence which utilizes monocular diplopia as a starting point. The interest in this fascinating phenomenon has, therefore, increased considerably of late; it has been taken out of the realm of purely academic problems, in which it had remained so long. Nevertheless, it still is by no means generally understood, and it would seem appropriate to discuss the phenomenon briefly in an attempt to elucidate it.

Monocular diplopia can occur only in patients who have an anomalous retinal relationship. But this anomalous correspondence must not be too deeply rooted; it must be sufficiently unstable to permit at least a temporary reappearance of normal correspondence.

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1. Miss Bischler (Bischler, V.: La triplopie binoculaire, Ann. ocul. **181**:724-732, 1948) believes that some insurmountable obstacle to binocular vision, such as cyclotropia, is necessary for the spontaneous occurrence of binocular triplopia. This is undoubtedly true in some cases, but it cannot be the absolute rule, as evidenced, for instance, by the case of Bielschowsky's (von Graefes Arch. Ophth. **46**:143-183, 1898).

2. Cass, E. E.: Monocular Diplopia Occurring in Cases of Squint, Brit. J. Ophth. **25**:565-577 (Dec.) 1941.

3. Walraven, F.: ARC Series. Directions for Use of a Monocular Diplopia Technique and Slides for the Treatment of Anomalous Correspondence, Columbus, Ohio, The Wottring Instrument Company, 1950. See also Kramer, M. E.: Clinical Orthoptics: Diagnosis and Treatment, St. Louis, C. V. Mosby Company, 1949, p. 349.

Spontaneous monocular diplopia will be found in patients in whom there is a rivalry between normal and anomalous correspondence. This will be the case either when anomalous correspondence is first established, at the onset of the strabismus, or when normal correspondence tries to reassert itself, for instance, after the eyes have been straightened surgically. The physician almost never has the opportunity to examine a patient in the very first stages of strabismus, or, if he does, the patient is ordinarily too young to permit a study of his visual act. Monocular diplopia is, therefore, most frequently seen in the postoperative stage, but even then it is rarely observed.

To elicit monocular diplopia artificially, one must present the patient with a stimulus situation which is unusual for him. This results in the reappearance of normal correspondence if the anomalous retinal relationship is not too well established and may in time lead to monocular diplopia. Such an unusual situation is created when the patient is forced to fixate with the habitually deviated eye. Javal,[4] who discovered monocular diplopia, described a patient with postoperative divergent strabismus who localized abnormally when fixating with his right eye. When he fixated with the habitually deviated (left) eye, he localized normally, and if a red glass was placed in front of that eye he perceived two images of a fixated candle, one red and two white ones.[5] In the unique and famous case of Bielschowsky's,[6] which started him on his career as an authority in neuromuscular disturbances of the eyes, an 18-year-old patient with monocular esotropia of his highly amblyopic left eye was compelled to fixate with this eye after the accidental loss of his normal (right) eye. Immediately after the accident the patient began to see double with his left eye, a condition which persisted for some time.

It is unnecessary to cite from the literature further examples of the occurrence of a change in the mode of localization with change in fixation, which may or may not be accompanied by monocular diplopia.[7] It should be emphasized, however, that the technique for the orthoptic treatment of anomalous correspondence advocated by Walraven consists essentially in the use of very small haploscopic targets presented in a major amblyoscope in such a way that the patient is urged or forced to fixate with the habitually deviated eye.

In the light of our knowledge of the visual act in strabismus, this is a very logical procedure. In this procedure the patient is first taught to fixate alternately with each eye, the haploscope arms being placed beyond the patient's subjective angle, i. e., in a divergent position as a rule. When alternate macular fixation is assured, the arms of the instrument are gradually converged until the subjective angle is reached, care being taken that the patient continues to fixate with the habitually deviated eye. At the subjective angle this becomes difficult for the patient, but this is also the point at which he should be asked to look for a third

4. Javal, E.: De quelques phénomènes de diplopie chez certains strabiques, Ann. ocul. **54**:123, 1865.

5. Javal, E.: Manuel du strabisme, Paris, G. Masson, 1896, p. 286.

6. Bielschowsky, A.: Über monoculäre Diplopie ohne physikalische Grundlage, nebst Bemerkungen über das Sehen Schielender, von Graefes Arch. Ophth. **46**:143-183, 1898.

7. For further details, see Burian, H. M.: Sensorial Retinal Relationship in Concomitant Strabismus, Arch. Ophth. **37**:504-533 (April) 1947.

image. If the patient perceives such a third image, he has monocular diplopia. This image is localized normally and appears at first dim and fuzzy and its colors are pale. Frequently this dim image is intermittently suppressed. It is now the task of the orthoptist to encourage the patient to concentrate attention on this dim image. At the same time the arms of the synoptophore are set closer and closer to the objective angle. The normally localized image gains in brightness, and the abnormally localized image eventually disappears.

We wish to cite briefly, as an example, the history of a patient with monocular diplopia whom we had the opportunity to observe.

REPORT OF A CASE

B. G., a 9-year-old girl,[8] was seen for the first time on July 28, 1950, with a history that the right eye had turned in since the age of 4 years. One brother had been operated upon for strabismus. The patient had worn glasses since 1945, according to the mother without improvement in the deviation. Refraction and visual acuity were as follows:

Right eye: +3.50 sph. $\bigcirc$ —0.50 cyl., ax 180 = 20/15

Left eye: +4.00 sph. $\bigcirc$ —0.50 cyl., ax 180 = 20/25

The prism and cover test gave the following results: with glasses: 40△ esotropia with 4△ right hyperphoria for distance and 55△ esotropia with 4△ right hyperphoria for near; without glasses: 65△ esotropia with 4△ right hyperphoria for distance and near.

The localization in the after-image test was normal, and in the double-image test the patient showed normal correspondence with the red glass over the left eye and anomalous retinal correspondence with the red glass over the right eye. On the synoptophore, the patient had an objective angle of 40△ base out and 3△ of right hyperphoria with crossed diplopia, but she could get momentary superimposition at that angle with small targets and stimulation. The subjective angle varied from 20 to 25△ base out with intermittent suppression of the left eye. When fusion targets were used, the patient could fuse at the objective angle with intermittent stimulation.

The patient's eyes were alternately patched, and she was given 16 sessions of orthoptic treatment, at the end of which she showed a somewhat unsteady normal correspondence on the synoptophore, was able to fuse small targets at the objective angle, and had some amplitudes.

In January, 1951, the patient was operated upon, with good cosmetic result. With glasses she had for distance a residual 5 to 7△ of esotropia with slight right hyperphoria. On the synoptophore her objective angle was 5△ base out; the subjective angle was not definite. The patient had postoperative orthoptic training, and on the seventh session, on Feb. 21, 1951, she observed an interesting phenomenon when small objects were used as targets on the synoptophore. These targets consisted of a ball 5 mm. in diameter, seen with the right eye, and a square of 9 mm., seen with the left eye. On the day of that examination the objective angle was zero, and the patient reported that she noticed two balls, a fuzzy one within the square and a bright one to the left of the square.

The child was then treated with the Walraven technique, and after six treatments she no longer had monocular diplopia with first- or second-grade targets; her objective and subjective angles coincided. However, in the after-image test she continued to show both normal and anomalous localization; in the diplopia test there was monocular diplopia with the red glass over the right eye; with the red glass over the left eye, she reported as a rule first crossed, then uncrossed, diplopia.

Orthoptic treatment of the patient is being continued. As tested on the machine, her binocular cooperation is improving, but the fusional amplitudes are still limited.

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8. This patient was referred to one of us (N.M.C.) for orthoptic treatment by Dr. Irving Pavlo, of Boston, who has permitted us to publish the case.

COMMENT

The case of this little girl is fairly characteristic. A child with a slight residual postoperative angle of squint presents monocular diplopia on the synoptophore. The normally localized image is at first dim; its colors are pale. As time goes by, and with the assistance of training, this fuzzy image gains in brightness, while the anomalously localized image grows dimmer until it is finally no longer observed once normal correspondence is fully established.

The difference in brightness between the two images seems to us to be an important factor in the explanation of the phenomenon; it is invariably reported in the cases described in the literature.

We wish to emphasize particularly that the monocular diplopia is due to the fact that in these cases the retinal elements have two spatial values which are simultaneously brought to consciousness. It is often said that one of the two images comes from an extramacular, the other from the macular, area. Accordingly, they are spoken of as the "macular" and the "extramacular" image. To think of monocular diplopia in this way is erroneous. The two images should be designated as the normally and the abnormally localized image.

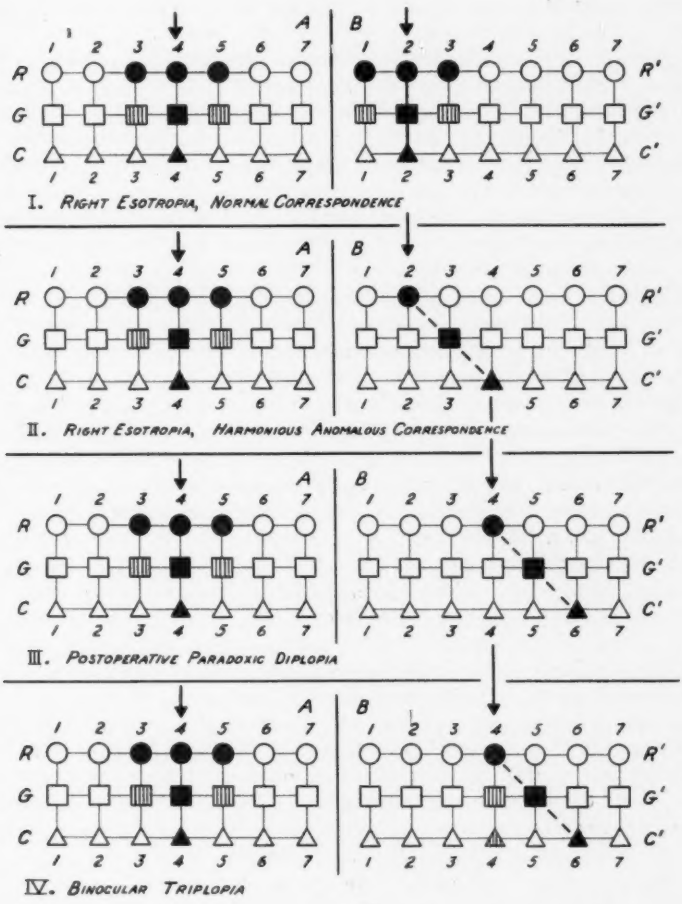
After a patient has been operated upon and his eyes are straightened, he often shows what is known as paradoxical diplopia. This simply means that he has kept his abnormal retinal relationship and that his two foveas maintain the two different visual directions which they had prior to the operation. In other words, he still shows the same abnormal localization which was present prior to operation when the arms of the major amblyoscope were set at the so-called objective angle of the patient. Before the operation the two foveas of the patient are stimulated when the arms of the synoptophore are set at this objective angle. After the operation, and if the angle of squint has been reduced to zero, the two foveas are again stimulated when the arms of the synoptophore are at the angle of zero. In neither case does the stimulus ever reach the extramacular area of the deviated eye on which the fixation point is imaged in casual seeing.

If the dormant normal correspondence can be reawakened in such a patient, he may simultaneously present normal and anomalous localization. This means that the foveal image of one eye is localized at the same time in two subjective visual directions, namely, paradoxically (crossed diplopia in esotropia) and normally, i. e., in the common visual direction of the foveas. Typically, the normally localized image is at first fuzzy, indistinct, and weak. As time goes by, or as treatment is given, the abnormally localized image becomes less brilliant, until it finally vanishes, while the subjective intensity of the normally localized image increases correspondingly. This transition does not have to be gradual; it may be rather abrupt.

The whole process may be expressed in neurophysiologic terms. As has been stated elsewhere,[9] the possibility of acquiring an abnormal retinal correspondence is preformed in the normal visual mechanism. When stereopsis occurs in the normal person, the fusion of disparate retinal images leads to an assimilation of visual directions. A similar, but much cruder, assimilation takes place in abnormal retinal correspondence.

9. Burian, H. M.: Scope and Limitations of Orthoptics in the Nonsurgical Treatment of Ocular Deviations, A. M. A. Arch. Ophth. **45**:377-381 (April) 1951.

In the accompanying chart, the situation is schematically represented.[10] The left side (*A*) symbolizes the left half of the visual system; the right side (*B*), the right half. The letters *R*, *G*, *C*, and *R'*, *G'*, *C'* indicate the left and right retina, geniculate body, and cortical areas, respectively.



Neurophysiologic explanation of anomalous correspondence and binocular triplopia. For explanation see text.

10. It should be clearly understood that this chart is a purely schematic presentation, intended to clarify the text. It is not meant to imply that the shift to be discussed actually takes place within the retina or the geniculate body, although this possibility is not excluded, nor should "cortical area" be interpreted as indicating area 17. It is entirely conceivable that the shift actually takes place in area 19.

All elements in these areas are closely interrelated, owing to the extensive arborization in the retina and the visual pathways; but it is assumed from Lorente de Nò's [11] studies that physiologically preformed preferential pathways exist which make possible a point-to-point relationship between retinal and cortical elements. Thus, a stimulus reaching the retinal area R_4 (left half of the figure), representing the left foveal area, is funneled, as it were, to the cortical area C_4, the central representation of the fovea of the left eye.

This mode of transmission of the excitation applies not only to the fovea, but to any area of the retina. If a patient has right esotropia, the image of the fixation point will fall in the right eye on an area nasal to the fovea. This retinal area is assumed to be R'_2 in part I of the chart, and the arrow indicates that this particular retinal area is stimulated. If the patient has normal correspondence, the retinal area R'_2 continues to transmit its impulses normally to the cortical area C'_2. Simultaneous stimulation by the fixation point of areas R_4 and R'_2 must therefore produce diplopia unless the image belonging to the right eye is suppressed, since the elements C_4 and C'_2 carry different local signs.

The retinocortical point-to-point relationship is a physiologic, not a rigid anatomic, one. The possibility exists that, through changes in threshold at the synapses, a given retinal area may come into physiologic relationship with any other cortical area. This makes anomalous correspondence possible.

Part II of the chart represents schematically a patient with right esotropia and anomalous correspondence. On the left side, the stimulus (arrow) falls on the retinal area R_4, which represents the left foveal region and transmits its impulses to the cortical region C_4. On the right side, the stimulus falls on the nasal retinal area, R'_2, but this retinal area is transmitting its excitations not to cortical area C'_2 but, rather, to cortical area C'_4.

It is assumed that the threshold of the pathway from R'_2 to C'_2 is raised and that there is a facilitation in the pathway from R'_2 to C'_4 because the image of the fixation point constantly impinges upon the extrafoveal area R'_2 when both eyes are open. Now C'_4 is the cortical region normally receiving stimulations from the foveal area R'_4. The stimulation of the cortical area C'_4 gives rise to a sensation of direction which corresponds to the sensation of direction produced by stimulation of C_4, on the opposite side; in other words, C_4 and C'_4 have the same local sign, and the stimulation of C'_4 results therefore in a directional localization of the stimulus which is identical with the direction in which stimuli to the foveal area on the opposite side are localized. Consequently a patient with right esotropia and anomalous correspondence said to be adapted to the angle of squint will see a stimulus falling on the retinal areas R_4 (left) and R'_2 (right) in one and the same visual direction.

If the two foveas of such a patient are now simultaneously stimulated, the result will be as shown in part III of the chart. On the left side the stimulus reaching R_4 will be transmitted to C_4, but on the right side the stimulus reaching R'_4 will be transmitted to C'_6. The stimuli reaching the two foveal areas are localized in two different directions. This situation will obtain preoperatively when the arms of the synoptophore are set at the patient's objective angle, and postoperatively if the patient's eyes are straightened.

11. Lorente de Nò, R.: Studies on the Structure of the Cerebral Cortex: II. Continuation of the Study of the Ammonic System, J. Psychol. u. Neurol. **46**:113, 1934.

In a patient with unstable anomalous correspondence, especially in a postoperative patient, one may now observe the phenomenon of monocular diplopia, which is schematically represented in part IV of the chart. The eyes have been straightened, and a normal stimulus situation has been achieved; i. e., the image of the fixation point falls now on the two foveas, as in a normal person. This may lead to reawakening of the normal correspondence. In the light of our neurophysiologic interpretation, this means that the transmission of the impulses from R'_4 to C'_4 is being facilitated. The impulses may, however, still go from R'_4 to C'_6. If the impulses are transmitted simultaneously from R'_4 to C'_4 and C'_6, this must lead to the localization of the one stimulus in two different directions at the same time. Obviously, the intensity of the excitation reaching C'_4 and the intensity of the excitation reaching C'_6 will not be equal. At first the intensity of the excitation reaching C'_4 will be low, and, accordingly, such a patient will perceive the normally localized image as fuzzy, unclear, or weak. As the transmission from R'_4 to C'_4 is facilitated, and, accordingly, the transmission of the impulse from R'_4 to C'_6 is inhibited, the intensity of the impulse reaching C'_4 will increase, and finally the impulse reaching C'_6 will become subliminal, with a corresponding increase of intensity of the excitation at C'_4. When this process is accomplished, anomalous correspondence has been replaced by normal correspondence.

It could be objected to this attempt at a neurophysiologic explanation of abnormal correspondence and binocular triplopia that it is nothing more than another way of describing the subjective phenomena. However, this would not be quite correct. It has been noted in studying the process of stereopsis in normal persons that no assimilation of the visual directions occurs when disparate retinal elements and their corresponding centers are simultaneously stimulated.[12] In other words, the stimulation of disparate retinal elements cannot be transmitted to the pathways coming directly from the retinal elements representing the corresponding centers while these retinal elements themselves are being stimulated. This indicates that the thresholds of the various synapses concerned with the transmission of the optical stimulations depend upon the condition of stimulation of neighboring synapses. Furthermore, the fact that the normally localized second image in monocular diplopia is at first fuzzy and indistinct is well explained by the above hypothesis. One would conclude, therefore, that the description given here is more than a simile and that it corresponds to actual occurrences within the central nervous system.

SUMMARY

Monocular diplopia (binocular triplopia) in cases of concomitant strabismus can exist only if the patient has an unstable anomalous correspondence, with resulting rivalry between the normal and an anomalous mode of localization.

Monocular diplopia is rare as a spontaneous occurrence but can relatively frequently be elicited artificially, e. g., when the patient is forced to observe haploscopic targets, fixating with the habitually deviated eyes. The patient then sees three images, two from the fixating eye, one from the other eye. One of the two monocular images is localized according to anomalous correspondence and appears bright and clear; the other is localized normally and appears at first faint and dim.

12. Burian, H. M.: Stereopsis, Docum. ophth., to be published.

The orthoptic technique for the treatment of anomalous correspondence developed by Walraven, utilizing these phenomena, is briefly described, and a case in point is reported in some detail.

It is pointed out that monocular diplopia is due to the fact that in cases of unstable anomalous correspondence the stimulated retinal elements have two spatial values which are simultaneously brought to consciousness. It is wrong to think that these two images come from two different retinal areas, one macular, the other extramacular. The two images should be designated not as the "macular" and the "extramacular" image, but as the normally and the abnormally localized image.

An attempt is made to give a neurophysiologic explanation of anomalous correspondence and binocular triplopia.

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FAULTY DEPTH PERCEPTION CAUSED BY CYCLOTORSION

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IN A RECENT paper, Nicholls [1] reviewed the literature dealing with the relation of heterophorias to depth perception. This author concludes that, within fairly wide limits, heterophoria seems to have little influence on the acuity of stereopsis. This is certainly true, inasmuch as cyclotorsional movements of the eyes are not involved.

In this paper an attempt is made to show that, under certain circumstances, misinterpretations of space frequently occur owing to cyclotorsion, i. e., wheel-like rotations of the eyes about their lines of fixation (Ogle [2]). Cyclotorsion is normally performed in the interest of proper alignment of the eyes to provide binocular single vision and reliable distance discrimination of vertically located objects. Cyclomotions of the eyes for this purpose are called "cyclofusional movements." According to Ogle,[2] the most evident characteristics of the cyclofusional mechanisms can be described as follows: 1. Both eyes perform essentially equal, but opposite, cyclotorsional movements (Hofmann and Bielschowsky [3]). 2. Cyclofusional movements are limited entirely to the so-called Panum's fusional areas (Verhoeff [4]). 3. The cyclofusional amplitude is unaffected by vertical divergence of the eyes enforced at the same time (Herzau [5]). 4. Cyclofusional movements vary in amount and reaction time with the individual subject.

Point 1 is important with respect to the analytical treatment of measurements concerned with cyclotorsion; point 2 indicates that the innervations leading to cyclofusional movements are equally conveyed, and point 3 gives rise to the conjecture that cyclofusion arises through an independent muscular synergy (Ogle [2]).

Under normal conditions of life the image patterns forcing cyclofusions are strong. This is true during daytime in surroundings rich in details, especially vertical contours.

Individual values of cyclotorsion reported by Tschermak [6] range from 0 to 1.307 degrees of arc (in extreme cases, 7 degrees), and combined values obtained from

From the United States Air Force School of Aviation Medicine.

1. Nicholls, J. V. V.: Relationship of Heterophoria to Depth Perception in Aviation, Am. J. Ophth. **33**:1497 (Oct.) 1950; **33**:1891 (Dec.) 1950.

2. Ogle, K. N.: Researches in Binocular Vision, Philadelphia, W. B. Saunders Company, 1950.

3. Hofmann, F. B., and Bielschowsky, A.: Der Willkür entzogene Fusionsbewegungen der Augen, Arch. ges. Physiol. **80**:1-40, 1900.

4. Verhoeff, F. H.: Cycloduction, Tr. Am. Ophth. Soc. **32**:208-228, 1934.

5. Herzau, W.: Über das Verhältnis von erzwungener Vertikaldivergenz und Rollung bei der Fusion, Arch. Ophth. **122**:59-74, 1929.

6. Tschermak, A. V.: Beiträge zur physiologischen Optik: IV. Augenbewegungen, in Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, Vol. 12, Pt. 2, 1930, pp. 1001-1094.

both eyes range from 0 to 4.85 degrees of arc (14 degrees in the extreme, according to Sachs and Meller [7]). Extorsion of 11.6 degrees and intorsion of 4.1 degrees were reported by Ames [8] as having been overcome.

Methods for determining existence of cyclotorsion are not numerous. Objective measurements of cyclomotions of the eyes, for instance, by applying a telescope and distinctive markings,[2] are uncomfortable and unreliable, and the accuracy has proved to be less than 0.5 degree of arc. With great accuracy, meridional afocal magnification lenses mounted in a geared unit were applied by Ogle. To avoid cyclofusional stimuli from the experimental device itself, the same author found that two points of light separated vertically could be used. On this principle the method employed in the present study was based. Its precision depends on the individual threshold of depth discrimination, whereby an angle of binocular parallax of 4.5 sec. corresponds to a cyclotorsion of about 0.1 degree of arc.

The problem in the present study was (1) to investigate the range and frequency distribution of cycloincongruities within a normal group of persons and (2) to study factors which are likely to evoke cyclotorsion.

METHOD AND PROCEDURE

A detailed description of apparatus and method used in this study has recently been given.[9] In brief, two round disks of equal brightness were separated vertically in the objective frontoparallel plane and observed in symmetrical convergence by the subject. Their angular size amounted to 5.7 min. of arc; their brightness, to 1.0 millilambert (mL.). The distance between the two objects was seen at a visual angle of 1.86 degrees of arc. In all the experiments, the subject was in a dark room and was required to make a depth judgment concerning the two illuminated disks while the upper target was seen with the naked eye and the lower one through a pair of plane-parallel plates of definite properties (Rollet's plates [10]). By contrarotating the Rollet plates about separate vertical axes, the apparent distance of the lower target could be varied optically with reference to the upper one. The test was simply to judge when the two disks were in the same apparent frontoparallel plane. Actually, the numerical reading was taken as the average of two readings, one corresponding to a judgment based on a clockwise rotation of the plates and the other to a judgment based on a counterclockwise rotation (method of limits). Then the average midpoint and the dispersion around the former one were determined.

Several experiments were performed on each subject. No measurements were made until a dark-adaptation period of 10 minutes had elapsed. Five preliminary measurements were then made but not used, thus allowing for the diminution of any learning effect. Generally, 10 average readings were made of the angular deflection required to achieve optically the plane coincidence of the apparent targets in each experiment. The average midposition (Z) and dispersion (σ^2) of settings were used as matter for the statistical disclosure of cycloincongruities or cyclotorsion. If the deviations (Z) from the expected midposition ($\bar{Z} = 0$) were definite in direction and significant statistically, they were regarded as evidence for cycloincongruities. The data obtained

7. Sachs, M., and Meller, J.: Über einige eigentümliche Lokalisationsphänomene in einem Falle von hochgradiger Netzhautincongruenz, Arch. Ophth. **57**:1-23, 1904.

8. Ames, A., Jr.; Cyclophoria, Am. J. Physiol. Optics **7**:3-38, 1926.

9. Cibis, P. A.; Wilson, M. R., and Fleck, H. G.: Studies on Effects of Windshields and/or Air of Different Densities on Stereoscopic Vision: II. Description of a Stereo-Meter and Method for Measuring Cyclo-Incongruities, Report No. 2, School of Aviation Medicine, Randolph Field, Texas, 1950.

10. Rollet, A.: Physiologische Versuche über binoculares Sehen, angestellt mit Hilfe planparalleler Glasplatten, Sitzungb. K. Acad. Wissensch. Math.-naturw. Klasse **42**:488-502, 1860.

by the method of limits were converted to angles of binocular parallax (ϵ), i. e., the angular disparity at which the targets appeared equidistant, expressed in seconds of arc. In the diagrams given for illustrating the individual behavior of eyes under certain conditions, the angular disparities (ϵ) were plotted as ordinates against time, expressed in minutes. By multiplying the angle ϵ by the conversion factor 0.022, the corresponding amount of cyclotorsion can be obtained in degrees of arc.

Selection of Subjects.—In the group of subjects given Tests 1, 2, 3, 4, and 5, which are described below, a minority had small errors of refraction. The majority had a visual acuity of 20/20 or better, none having less than 20/30 vision in the poorer eye. In the group of subjects given Tests 6, 7, and 8, the visual acuity of both eyes was at least 20/20 without glasses. Refractive errors were generally corrected during the performance of tests. All but one subject taking Tests 1, 2, and 5 had passed the Howard-Dolman test (disparity tolerance, 11.3 sec. of arc). Patients with heterotropia and suppression were eliminated. About 80% of the observers were between 16 and 25 years of age; the rest, not older than 50.

It is desirable to know whether the average deflection in Test 1 differs from that of Test 2, and similarly for Tests 3 and 4.

TESTS AND MEASUREMENTS

Tests 1 and 2.—The test objects were presented in a dark room at an observation distance of 6 meters. In Test 1 no stimuli for cyclofusion existed, whereas in Test 2 the most proximal vicinity of the targets was illuminated with 2.6 foot-candles (f.-c.), providing perceptibility of the contours of a rectangular frame in upright position, and therewith cyclofusional compulsions. The brightness of the frame amounted to 0.25 mL.; that of the targets to 1.0 mL. The side of the frame was 16 in. (40.6 cm.) in length. A total of 30 subjects were examined. The mean score of the thresholds of binocular parallax at which the targets appeared amounted to 10.07 ± 3 sec. of arc in Test 1, to 9.07 ± 2.6 sec. of arc in Test 2, and to 5.20 ± 8.3 sec. of arc in the Howard-Dolman test. The analysis of variance technique [11] disclosed that the tests are significantly different ($P<0.001$). To determine the comparisons which contributed to this result, a t test between each pair of tests was performed. The results are shown in the following tabulation:

Tests Compared	t	df	P
1; 2	1.029	29	0.3
1; Howard-Dolman	4.758	29	0.01
2; Howard-Dolman	2.948	29	0.01

These values showed that the three tests did not yield the same results, and that the results for Tests 1 and 2 differed from those for the Howard-Dolman test, but not from each other. Therefore, the Howard-Dolman test was not further considered.

By means of the measurements obtained in the single tests, the angular disparity (ϵ) at which the sagittal-distance difference between the apparent targets is seen, and which is caused by cyclotorsion or cycloincongruities, can be computed, and in like manner also the totaled amount of cyclotorsion, which may be designated as δ, will be given in degrees of arc.

In Chart 1 the frequency distribution of the grouped data obtained in Tests 1 and 2 is shown after conversion into ϵ and δ. No information about the significance of the deflections of the individual midpositions from the zero position can be

11. Dr. Max Halperin, former Chief of the Department of Biometrics, USAF School of Aviation Medicine, Randolph Field, Texas, made the statistical computations.

gained from the histograms in Chart 1. Therefore, the following considerations have been made:

On the basis of assumptions outlined in the appendix, one can carry out an F test for difference in means (or, equivalently, a t test). For Tests 1 and 2, in which 30 subjects were used, the results are summarized in Table 1.

According to Table 1, using a 0.05 significance level, 22 of the 30 subjects gave evidence of a marked discrepancy in average deflection between Tests 1 and 2.

TESTS 3 and 4.—A similar procedure was applied in these tests. Both tests were performed at an observation distance of 3 meters, Test 3 being carried out in the completely dark room. In Test 4 the target environment was also kept completely dark, but a white-lined adaptation globe with a square outlet on the back

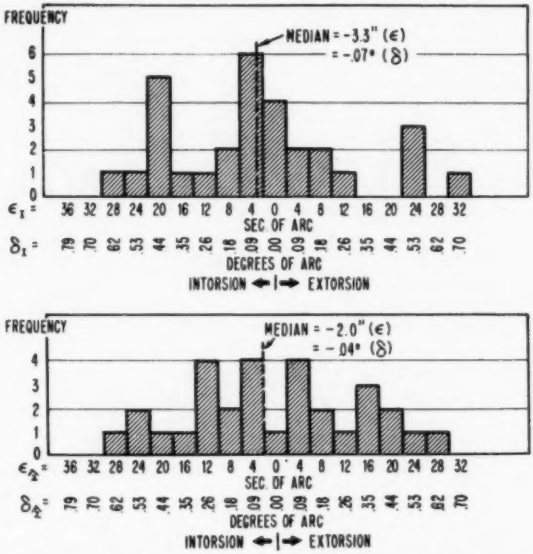

Chart 1.—Frequency distribution of the averaged and grouped data obtained for the deflection of the midpositions from the expected position in Tests 1 (above) and 2 (below). The ϵ scales exhibit the angular disparities (angles of binocular parallax); the δ scales, the total amount of cyclo-incongruities (cyclotorsions plus anomalous relationship of the two retinas).

TABLE 1.—*Number of Determinations (Subjects) at Different Probability Levels*

Probability level	Less than or equal to 0.001	Less than or equal to 0.01	Less than or equal to 0.05	Greater than 0.05
Number of determinations	16	20	22	8

provided an illumination of 260 f.-c. at the entrance pupil. The outlet was made large enough to avoid obstruction of the visual lines. These applications yielded bright adaptation and cyclofusional compulsions. The results of Tests 3 and 4 for 70 subjects are shown in Chart 2 and Tables 2 and 3.

According to Table 3, using a 0.05 significance level, 48 of the 70 subjects gave evidence of a marked discrepancy in an average deflection as between Tests 3 and 4.

Since it was impossible to obtain very thorough information about the individual behavior of the eyes with respect to cyclofusional movements under different experimental conditions from statistical material, further experiments were performed with subjects who were distinguished by a high degree of reliability in preliminary tests given for this purpose.

TEST 5.—After bright adaptation to 260 f.-c., lasting for 2 to 10 minutes, the subjects made depth judgments concerning the two transilluminated disks during a subsequent period of darkness in the laboratory. In many cases periods of brightness and darkness changed alternately several times. The data obtained by the

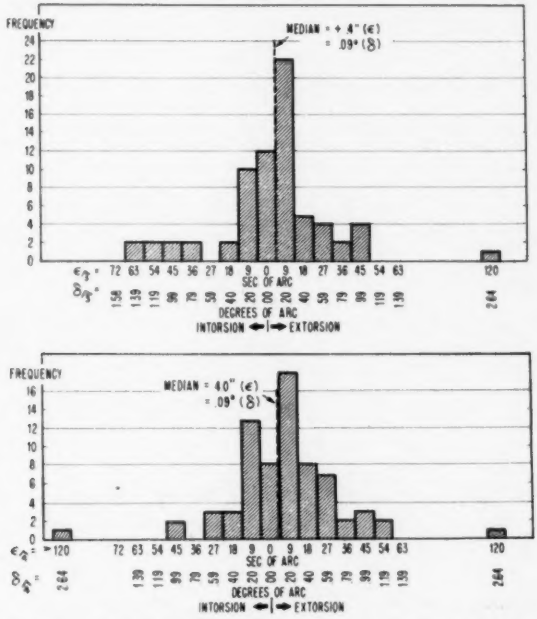

Chart 2.—Frequency distribution of the averaged and grouped data obtained for the deflection of the midpositions from the expected position in Tests 3 (above) and 4 (below). Further explanation is given in the legend to Chart 1.

method of limits were converted to those of angular disparities (ϵ). They were plotted then on the ordinate against the time, in minutes. The angular disparity can be considered a measure of the cyclomotional deviation from the true, or expected, position. It may be recalled that by multiplying the angle ϵ by the conversion factor 0.022, the corresponding amount of cyclotorsion can be obtained in degrees of arc. The dispersion of the measurements is given by the blackened bands in Chart 3*A* and the charts following; their deflection from the true, or expected, values, by their deviation from the zero level. The positive angle ($+\epsilon$) refers to extorsion; the negative angle ($-\epsilon$), to intorsion.

The 19 subjects tested seem to fall into three distinct groups: The first group comprises about one-third of the examinees and is distinguished by a notable constancy and precision of the measurements under the conditions encountered in Test 5.

The second group is distinguished by a good or fair precision of the measurements, but by remarkable shifts of the averaged midpositions from the zero level

TABLE 2.—*Frequency Distribution of Deflection of Midpositions from Zero Position, Measured in Units of Standard Deviation (σ).*

Test 3

		$-\sigma s$											$\pm$	$+\sigma s$											Total
		30	20	10	9	8	7	6	5	4	3	2	1	<1	1	2	3	4	5	6	7	8	9	10	20	30	Number
$+\sigma s$	60	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	50	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	1
	40	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	30	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	2
	20	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	10	..	..	..	..	..	..	1	..	..	..	..	..	..	..	1	1	1	..	..	..	..	2	..	..	6
	9	..	..	..	..	..	..	..	..	..	..	..	..	..	..	2	..	..	..	..	..	..	..	..	..	2
	8	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	7	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	6	..	..	..	..	..	..	..	..	..	..	..	1	1	1	..	..	..	..	..	..	..	..	..	..	2
	5	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1
	4	..	..	..	..	..	..	..	..	..	..	..	..	..	2	1	..	..	..	..	..	..	..	..	..	3
	3	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	1
	2	..	..	..	..	..	..	..	..	..	..	..	4	2	..	1	..	..	..	..	..	..	..	..	..	7
	1	..	..	..	..	..	..	..	..	..	..	..	6	4	1	..	..	..	..	..	..	..	..	..	..	11
$\pm$	<1	..	..	..	..	..	..	..	..	..	..	..	8	2	2	..	..	..	..	..	..	..	..	..	..	12

Test 4

$-\sigma s$	1	..	..	..	..	..	..	..	..	..	1	..	1	2	2	2	..	..	..	..	..	..	..	..	..	..	8
	2	..	..	..	..	..	..	..	..	..	..	..	..	3	..	..	1	..	..	..	..	..	..	..	..	..	4
	3	..	..	..	..	..	..	..	1	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	2
	4	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
	5	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	6	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
	7	..	..	..	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
	8	1	..	..	..	..	..	..	..	..	..	..	..	1	1	..	..	..	..	..	..	..	..	..	..	..	3
	9	..	..	..	..	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
	10	..	1	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
	20	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
	30	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..
Total		1	1	..	..	..	..	1	3	..	3	1	2	25	14	11	3	1	..	..	..	..	1	3	..	..	70

TABLE 3.—*Number of Determinations (Subjects) at Different Probability Levels*

Probability level	Less than or equal to 0.001	Less than or equal to 0.01	Less than or equal to 0.05	Greater than 0.05
Number of determinations	32	39	48	22

toward (*a*) the plus or (*b*) the minus side. The results of several subjects have been selected to illustrate these types. They are presented in Charts 3 to 5. From the inspection of the graphs in Charts 3*B* and 4, it is evident that cyclofusional compulsions can still be effective after the period of darkness is initiated, but they mostly diminish gradually and finally attain their lowest level after approximately 15 minutes. On the other hand, illumination of the adaptation globe is associated with an immediate shift of the trends mostly to the plus side, which is sometimes overbalanced in the first minutes. In Chart 4 graphs are displayed which were obtained from one subject in test and retest on three different days.

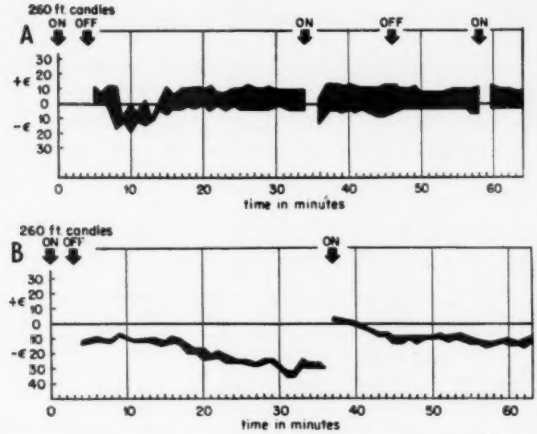

Chart 3.—*A* (Observer J. L.), representing a constant type which is not significantly affected by changes in the illumination.

B (Observer R. W.), representing a type with significant shifting of the midposition due to cyclotorsion, intorsion during darkness, and extorsion if 260-f.-c. illumination is switched on.

In Charts 3*A* and *B*, 4, and 5, settings of limits are given as angular disparities (ϵ) in seconds of arc plotted against time during brightness (*on*) or darkness (*off*). Round targets were vertically set. The blackened band represents the amplitude of depth discrimination. Significant deviations from the zero level denote cycloincongruities due to anomalous retinal correspondence or to cyclotorsion. Cyclotorsion was assumed if the shifting of the midposition from one position to another was evident.

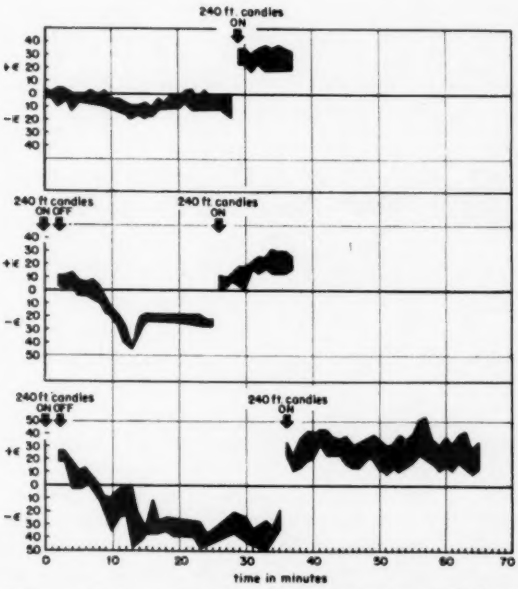

Chart 4.—Observer P. R. represents a type with extorsion at brightness and intorsion at darkness. The three tests were made on different days. Note that the first measurements shown in the diagram above were obtained without preceding bright adaptation.

The third group is distinguished by a more or less striking loss in depth discrimination when the objects are presented in a completely dark room.

TEST 6.—This test concerns the influences of oxygen deficiency on depth discrimination and cyclofusion. The experiments were performed in five steps, each lasting for 10 minutes, with breaks of 3 minutes between. In the first phase,

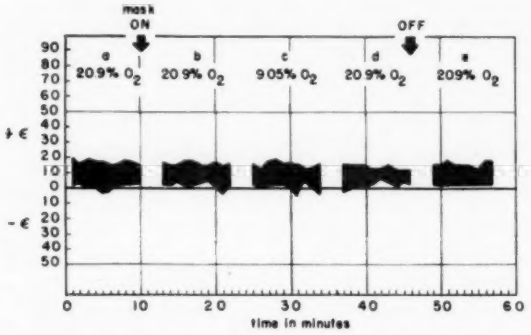

Chart 5.—Oxygen test. Observer D. E. represents a constant type with no significant deviations of the midpositions from the zero level.

In this chart, and in Chart 6, settings to limits were based on the criterion of apparent "equidistance" under different oxygen supplies.

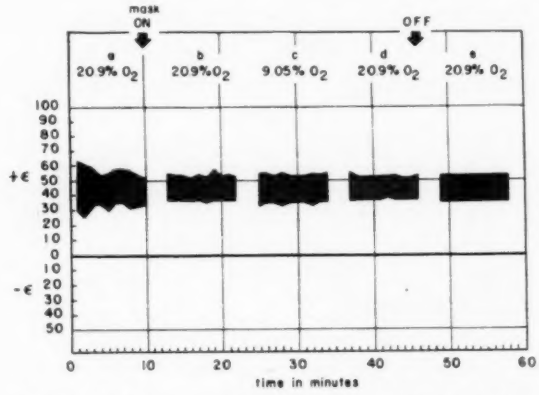

Chart 6.—Observer J. L. represents a constant type with significant, but constant, deviations (anomalous retinal correspondence[?]) of the midpositions from the zero level.

the subject breathed normal air without mask; in the second, normal air through the mask; in the third, a nitrogen-oxygen mixture of 9.05% oxygen content; in the fourth, normal air again with mask on, and in the fifth, normal air without mask. In this series of tests, also, the 15 subjects seem to fall into three distinct groups: The first is distinguished by a remarkable constancy and accuracy of the measurements in all phases (Charts 5 and 6); the second, by a small dispersion of

the measurements but notable shifts of the averaged midpositions from the primary level toward (*a*) the plus (Chart 7*A*) or (*b*) the minus (Chart 7*B*) side, and the third, by an increased inaccuracy of the measurements in oxygen deficiency (Chart 8). It may be noteworthy that subjects tested in Tests 5 and 6 showed the same behavior.

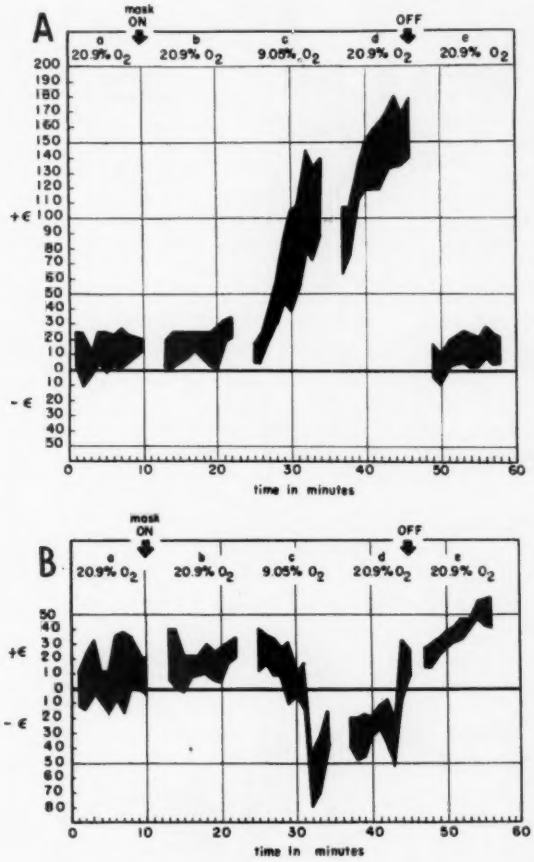

Chart 7.—*A* (Observer X. W.), significant extorsion in oxygen deficiency. *B* (Observer R. W.), significant intorsion in oxygen deficiency.

TEST 7.—This test concerns the effect of alcoholic intoxication on depth discrimination and cyclofusion. It was generally performed in two steps: First, measurements were made under normal conditions over a period of 10 minutes. Thereafter, the examinee consumed approximately 0.36 ml. of 100% alcohol per pound of body weight. The alcohol was mixed with soda water. Subsequent measurements were made in intervals over a period of several hours. Unfortunately,

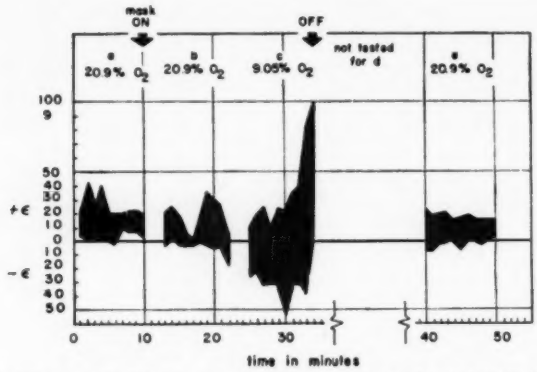

Chart 8 (Observer X. E.).—Increased amplitude of depth discrimination in oxygen deficiency.

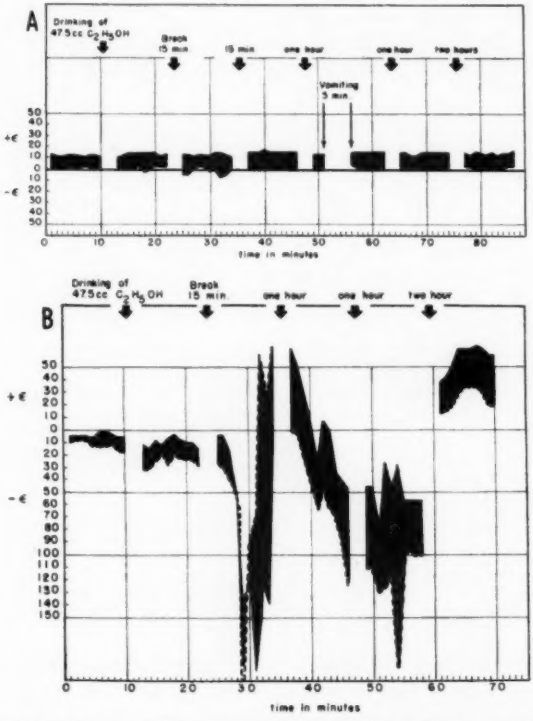

Chart 9.—Alcohol test. *A* (Observer D. E.), depth discrimination not affected by alcoholic intoxication.

B (Observer X. C.), significant effect of alcoholic intoxication. The notched margin represents settings to the limit of apparent "front" positions; the plain margin represents settings to the limit of apparent "rear" positions. Note the confusion (inversion) and inconstancy of the settings after consumption of alcohol.

only five volunteers could be examined, so that it is hard to make a general statement about the effect of alcohol on cyclofusion and depth discrimination. But from the data obtained, it is evident that the depth-discriminating ability of most of these subjects was not affected by the alcohol, nor was there any change in their cyclofusion ability (Chart 9*A*), although a high degree of intoxication was obtained (nausea and vomiting). Incidentally, all these subjects had also undergone Tests 5 and 6, and all fell into the first group, i. e., subjects showing constancy in the average midposition and dispersion of measurements. Charts 5 and 9*A*, for example, present measurements obtained from the same subject. On the other hand, one of the subjects showed a complete loss of spatial orientation as an effect of the alcoholic intoxication, as shown in Chart 9*B*.

CONCLUSIONS

From the experiments, certain basic conclusions can be drawn:

1. Cyclotorsion of the eyes takes place readily in everyday life.

2. About 60 of 100 persons show marked cyclotorsion as a result of changes in the environmental conditions.

3. There is a group of persons, roughly estimated between 30 and 40%, distinguished by great constancy and precision in judgment of depth, who cannot be affected by circumstances. The depth judgments of these persons spread either around the zero level or show a constant deviation in the averaged midpositions. This constant error is supposedly caused by cycloincongruities based on anomalous relationships of the two retinas.

4. Oxygen deficiency can cause discrepancies in the depth perception but does not affect the judgment of persons mentioned in paragraph 3.

5. Alcoholic intoxication does not cause cyclotorsion in persons who fall in the group with constant depth judgment, mentioned in 3.

APPENDIX

The statistical treatment of the data obtained in Tests 1 through 4 is based on the following considerations, proposed by Dr. Max Halperin:

It is reasonable to assume that each subject has a different average deflection and that the series of readings taken of the average deflection will vary around the "true" value according to a Gaussian (bell-shaped) curve. It is also reasonable that the precision of each subject will differ; that is, measurements of some subjects will more frequently be closer to their "true" values than those of certain other persons. Thus, one method of approach is to consider each subject as a universe and to take the samplings in each experiment as independent drawings from that universe. Knowing that the precision remains approximately the same for each subject in all experiments, we ask whether or not there is a difference in the "true" deflection as judged by comparison of the experimental results. Other approaches to the problem are possible, but the one described above was chosen for simplicity of analysis and because the question could be posed and answered in a rather meaningful way. On the basis of the assumptions outlined above, the F test for difference in means (equivalent to a t test) was carried out.

Considering the data obtained by statistical treatment as a whole, it may be assumed that the true average deflection and precision for each subject are themselves samplings from some bivariate superpopulation. Then, taking the differences in average deflections in the two pairs of experiments (Tests 1 and 2 and Tests 3 and 4, respectively), one would want to test the hypothesis that in the superpopulation of average differences so obtained the true average differences were zero.

Aside from the question of whether such a modeling of the situation is more or less realistic than the model actually used, the mathematical analysis of such a model becomes fairly complicated and has not been worked out in detail. When and if a technique for analysis according to such a model becomes available, it may be of value in such problems as the present one.

AN EXPERIMENTAL STUDY OF OPTOKINETIC RESPONSES

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A NORMAL person, as Walsh [1] and others have pointed out, will exhibit an optokinetic (or opticokinetic) nystagmus on watching a succession of stationary objects as he moves steadily past them, such as the telegraph poles seen from a moving train. Likewise, it may appear if his eyes follow a series of moving objects, such as the stripes on an optokinetic drum. A nystagmus of this type consists of a following movement, or a slow phase, and a quick return of the eyes to center, or a quick phase. The slow phase depends upon fixation of the eyes. Various ophthalmologists have stated that such a nystagmus may be avoided if the subject being tested will visualize some object beyond the optokinetic drum during the test (Walsh [1]) or will fix his attention on something other than the drum (Kestenbaum [2]). However, as will be seen, not all observers (Cogan and Loeb [3]) have agreed that it is possible to inhibit optokinetic nystagmus voluntarily.

Clinicians in general have believed that some ability to see, and so an at least partially intact visual cortex (Area 17), is necessary in order that optokinetic responses may occur; or, to state it as Walsh has done, a person who exhibits such a nystagmus on adequate testing is not totally blind. Opinions differ as to whether an intact macula is essential for such optokinetic responses. Fox and Holmes [4] and Dodge and Fox Jr.[5] believed that optokinetic responses may be elicited from the periphery of the retina. Kestenbaum [2] claimed that a response of this type is inter-

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1. Walsh, F. B.: Clinical Neuro-Ophthalmology, Baltimore, Williams & Wilkins Company, 1947, pp. 318-320.

2. Kestenbaum, A.: Clinical Methods of Neuro-Ophthalmologic Examination, New York, Grune & Stratton, Inc., 1946, pp. 221-230.

3. Cogan, D. G., and Loeb, D. R.: Optokinetic Responses and Intracranial Lesions, Arch. Neurol. & Psychiat. **61**:183-187 (Feb.) 1949.

4. Fox, J. C., and Holmes, G.: Optic Nystagmus and Its Value in the Localization of Cerebral Lesions, Brain **49**:333-371 (Sept.) 1926.

5. Dodge, R., and Fox, J. C., Jr.: Optic Nystagmus: I. Technical Introduction with Observations in a Case with Central Scotoma in the Right Eye and External Rectus Palsy in the Left Eye, Arch. Neurol. & Psychiat. **20**:812-823 (Oct.) 1928.

fered with when the macula lutea does not function because of pathological involvement or because it is not well developed, and that an image which shows no contours as it passes over the macula will not produce such an optokinetic response. He stated:

> For elicitation of the O.N. [optokinetic nystagmus] a central vision of at least finger counting in short distance is required. . . . If a central scotoma is so large that it includes the entire macula . . . optokinetic nystagmus is found absent or at least diminished.

Stimulation of the preoccipital eye field (Area 19) in primates (for example, in man by Foerster [6] and in the macaque by Crosby and Henderson [7]) and of the various portions of the occipital eye field (Area 18) in the macaque (Walker and Weaver [8]; Crosby and Henderson [7]) indicates that these brain regions are related to conjugate deviation of the eyes in various planes and that the pattern for such deviation is well localized on the cortex. It is rather generally conceded that these preoccipital and occipital eye fields are functionally related to following movements of the eyes or to the fixation of the eyes on a moving object or on one at rest. Consequently, if these cortical regions are involved in a lesion, the response to be expected, when the patient is tested by rotation of an optokinetic drum away from the side of the lesion, will be a deficiency in such a following movement, that is, in the slow, or following, phase of the optokinetic nystagmus. Cogan and Loeb [3] found that the following responses were defective in 58 of their clinical cases, in 57 of which the response was impaired when the drum was rotated toward the side of the lesion. They pointed out that such results were surprising in the light of the cerebral representation of ocular movements to the opposite side, and they were unable to explain the seeming inconsistency. These workers reported that abnormalities in optokinetic responses are more frequent when the lesion involves the posterior or middle portion of the cerebrum.

Various observers have suggested that regions of the cerebral cortex other than the occipital—such as the frontal lobe (Fox [9] and others), the angular gyrus (Bárány [10]), the supramarginal and angular gyri with adjacent portions of the parietal and temporal lobes (Fox and Holmes [4]), the deep white matter of the hemisphere (Cords,[11] Ohm,[12] Strauss,[13] and others), or brain stem centers (Scala

6. Foerster, O.: The Cerebral Cortex in Man, Lancet **2**:309-312 (Aug. 8) 1931.

7. Crosby, E. C., and Henderson, J. W.: The Mammalian Midbrain and Isthmus Regions: II. Fiber Connections of the Superior Colliculus: B. Pathways Concerned in Automatic Eye Movements, J. Comp. Neurol. **88**:53-91 (Feb.) 1948.

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9. Fox, J. C., Jr.: Disorders of Optic Nystagmus Due to Cerebral Tumors, Arch. Neurol. & Psychiat. **28**:1007-1029 (Nov.) 1932.

10. Bárány, R.: Zur Klinik und Theorie des Eisenbahnnystagmus, Acta oto-laryng. **3**:260-265, 1921.

11. Cords, R.: Optisch-motorisches Feld und optisch-motorische Bahn: Ein Beitrag zur Physiologie und Pathologie der Rindeninnervation der Augenmuskeln, von Graefes Arch. Ophth. **117**:58-113 (May) 1926.

12. Ohm, J.: Optokinetischer Nystagmus und Nystagmographie im Dienste der Hirndiagnostik, Arch. Augenh. **106**:185-220, 1932.

13. Strauss, H.: Über die hirnlocalisatorische Bedeutung des einseitigen Ausfalls des optokinetischen Nystagmus und der hemianopischen Aufmerksamkeitsschwäche, Ztschr. ges. Neurol. u. Psychiat. **143**:426-435 (Jan.) 1933.

and Spiegel [14] and others)—may have important functional relations to optokinetic responses. Thus, Bárány [10] believed that the quick phase of these responses may arise from the frontal lobe; the slow phase, from the angular gyrus.

From the data obtained from a study of two clinical cases of homonymous hemianopsia, Bárány [10] concluded that optokinetic nystagmus is lost when the optokinetic drum is rotated toward the side of the hemianopsia, but is present, in normal fashion, when it is rotated toward the other side. Ohm [12] pointed out that Bárány's statement is true only when the efferent pathway from the occipital cortex, as well as the visual radiations (his sensory visual path), is destroyed. Cords [11] and Strauss [13] emphasized strongly the importance of this efferent system from the occipital region to lower centers. They opposed Stenvers' [15] view (and that of Fox and Holmes [4]) that the frontal eye field is a necessary part of the efferent arc from the occipital cortex to brain stem centers. The efferent discharge paths (Beevor and Horsley [16]; the internal corticotectal tracts, preoccipital and occipital divisions, and the corticotegmental tracts of Crosby and Henderson [7]) from the appropriate regions of the cortex, to and through midbrain areas, are well known. The interference with vertical (and the sparing of horizontal) following movements of the eyes in cases of pinealoma (Crosby and Henderson [7]; Crosby [17]), in which appropriate portions of these paths are destroyed, stress the relations of these efferent systems to optokinetic nystagmus. In his review of the matter, Walsh [1] stated that inability to produce optokinetic nystagmus indicates, most frequently, that the efferent paths from the occipital lobes have been damaged.

Scala and Spiegel [14] found that lesions in the superior colliculi of dogs and cats block the optokinetic responses. Their results may have been due to an interruption of efferent (corticotegmental) paths from the preoccipital and occipital eye fields, since a considerable proportion of such paths (Crosby and Henderson [7]) course just beneath this brain area. It is highly probable, however, that in subprimates, in which a much larger percentage of the optic system terminates directly in the superior colliculi (Crosby and Henderson [7]), cortical participation in optokinetic responses is much less vital.

A very interesting study of optokinetic nystagmus has been presented by Droogleever Fortuyn and van der Waals,[18] who used optokinetic drums of different sizes, one of which was so constructed that the patient could be placed inside of it. They found that an extension of the revolving field of vision led to a reinforcement of the optokinetic stimulus. Moreover, in patients having focal lesions of the hemisphere, an asymmetrical optokinetic nystagmus could always be obtained on testing, provided a sufficiently strong stimulus was employed. When patients with definitive

14. Scala, N. P., and Spiegel, E. A.: Mechanism of Optokinetic Nystagmus, Tr. Am. Acad. Ophth. **43**:277-303 (Oct.) 1938.

15. Stenvers, H. W.: Über die klinische Bedeutung des optischen Nystagmus für die zerebrale Diagnostik, Schweiz. Arch. Neurol. u. Psychiat. **14**:278-288, 1924.

16. Beevor, C. E., and Horsley, V.: On the Pallio-Tectal or Cortico-Mesencephalic System of Fibres, Brain **25**:436-443, 1902.

17. Crosby, E. C.: Application of Neuroanatomical Data to the Diagnosis of Selected Neurosurgical and Neurological Cases, J. Neurosurg. **8**:566-583 (Nov.) 1950.

18. Droogleever Fortuyn, J., and van der Waals, H. G.: De optokinetische nystagmus bij lijders aan haardprocessen in het centrale zenuwstelsel, Nederl. tijdschr. geneesk. **84**:4602-4612 (Nov.) 1940.

pontine lesions were tested, no optokinetic responses to one side were obtained. If there was a disturbance in the optokinetic nystagmus, the eyes showed jerky movements when an attempt was made to follow an object moving across the field of gaze toward the other side, that is, away from the direction of the rapid, or return, component of the nystagmus.

Cogan and Loeb [3] stated that optokinetic responses cannot be inhibited voluntarily. Considered in this context, the studies on intracortical connections by McCulloch [19] present some interesting data regarding the relations existing between the eye fields of the frontal and those of the occipital and preoccipital regions. This observer reported that impulses from Area 8 (which falls within the frontal eye field) are excitatory over Area 18 (the occipital eye field) of both sides but suppress the contralateral and homolateral Areas 19 (which include the preoccipital eye fields). Area 18 does not fire Area 8 of either side, but Area 19 is a suppressor of Area 8 of the same and the opposite side. These results of McCulloch and those obtained from a study of the frontal eye fields of the macaque (Macaca mulatta) have led us (Crosby, Yoss, and Henderson [20]) to the conclusion that at least in these monkeys—and probably in other primates—the optokinetic responses which can be obtained from the occipital (Area 18) and preoccipital (Area 19) eye fields are under the regulatory control, or dominance, of the frontal eye fields. Consequently, experiments were planned to confirm or disprove this apparent dominance. These experiments and their results are reported in the following pages.

MATERIAL AND METHODS

An apparatus was constructed for testing the optokinetic response. This consisted of a large box (Fig. 1), in which the monkey could be placed after being immobilized with the head in a stereotaxic apparatus (McCulloch modification). The head position was such that the eyes were on a horizontal plane. An opening was made in the box directly in line with the animal's gaze and of such dimensions that it could be filled by a rotating optokinetic drum (15.9 cm. in diameter; 15 cm. in height) with vertical stripes (12 mm. wide). Viewing windows were fitted with one-way mirrors, so that the observer could watch the ocular responses without detection. There was an electric bulb in the ceiling of the box, and the outer room was darkened in order to make the mirrors effective.

It was necessary to anesthetize each animal lightly with ether in order to place him in the apparatus. Readings were taken after the animal became conscious and with complete silence maintained by the observers in order to avoid distracting impulses. The interior of the box was painted a dull black for the same reason.

Several of the monkeys used in this study of optokinetic nystagmus were also employed in determining the localization pattern on the frontal eye fields (Crosby, Yoss, and Henderson [20]). Consequently, in some of the animals, portions of the frontal eye fields were destroyed during the course of these experiments. In the same, or other, animals the preoccipital and/or the occipital eye fields were ablated. The order of surgical intervention varied from monkey to monkey. In each animal the optokinetic responses in the horizontal plane were tested both to the right and to the left before any operative procedure. The tests were repeated after each operation in the series of experiments performed on each animal. The surgical procedures were performed with the animal under ether anesthesia. The skull was trephined, and the area to be destroyed was exposed and ablated by cauterization. Wherever possible, the dura was closed

19. McCulloch, W. S.: Corticocortical Connections, in Bucy, P. C., Editor: The Precentral Motor Cortex, Ed. 2, Urbana, Ill., University of Illinois Press, 1949, Chap. VIII, pp. 211-242.

20. Crosby, E. C.; Yoss, R. E., and Henderson, J. W.: The Mammalian Midbrain and Isthmus Regions: II. The Fiber Connections: D. The Pattern for Eye Movements on the Frontal Eye Field and the Discharge of Specific Portions of This Field To and Through Midbrain Levels, J. Comp. Neurol., to be published.

over the area; the connective tissue and skin were approximated. Sterile technique was maintained throughout. The animals were allowed to survive for varying lengths of time in order that the optokinetic testing might be continued. Most of the operations were additive in type, successive cortical areas being removed.

It is to be stressed that the aim of this series of experiments was not to produce a maximum optokinetic nystagmus. The presence of such a nystagmus on proper stimulation is generally recognized. It was, rather, to use a stimulation just adequate to arouse an optokinetic response when the monkey's eyes were directed toward the stripes of the drum, but still not so dominant but that he could suppress such optokinetic movements voluntarily. Such suppression was attained when the monkey, through fear and curiosity, directed his attention to other matters, rather than to the stripes on the drum, even though he was looking directly toward these stripes (although probably not actually focusing upon them). Thus, facilitation of the occipital and preoccipital areas by the visual stimuli was played over against their suppression (or inhibition) by the frontal eye fields. Such an experimental set-up having been established, the effects of frontal and preoccipital and occipital ablations on the optokinetic responses were tested.

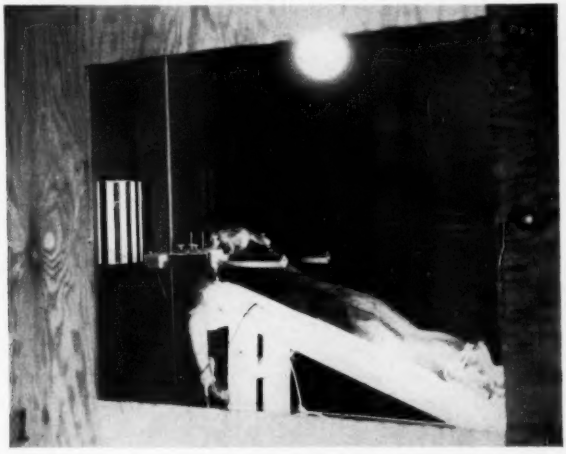

Fig. 1.—Monkey placed in stereotaxic apparatus. The optokinetic drum and one viewing window are shown. The opening through which the picture was taken has a sliding door.

ANATOMICAL CONSIDERATIONS

Before the results of the experiments are reported, it has seemed advisable, as a matter of convenience for the reader, to review briefly the localization patterns on the preoccipital (Area 19), the occipital (Area 18, possibly part of Area 17), and the frontal eye fields in the macaque, since an evaluation of these results depends to a great extent upon having such patterns clearly in mind. The localization patterns here reviewed in very brief fashion are those reported for the preoccipital and occipital eye fields by Crosby and Henderson[7] and for the frontal eye fields by Crosby, Yoss, and Henderson.[20] Those interested in the evidences for these patterns and in the pertinent literature, to which many have contributed, are referred to the original articles, together with their bibliographies.

Discrete stimulation of the preoccipital eye field (Fig. 2), beginning at its upper border and proceeding downward over the midlateral surface of the lateral hemi-

sphere wall of the macaque, produced the following conjugate deviations of the eyes: (*A*) eyes turned upward (sometimes slightly obliquely upward); (*B*) eyes directed obliquely upward toward the side opposite the stimulation; (*C*) horizontal deviation of the eyes away from the side stimulated; (*D*) eyes directed downward and toward the side opposite the stimulation; (*E*) eyes turned downward (or sometimes slightly obliquely downward).

From the upper part of the occipital eye field (Fig. 2*D'*) in the macaque, electrical stimulation produced a conjugate deviation of the eyes downward and toward the side opposite stimulation, a reaction comparable to that obtained on stimulation of the lower part of the preoccipital eye field (Fig. 2, *D*). Stimulation of the lower part of the occipital eye field (Fig. 2, *B'*) in the macaque resulted in an obliquely upward conjugate deviation of the eyes, with their slant away from the side stimulated. This result had been obtained earlier by Walker and Weaver.[8]

From these occipital and preoccipital regions, corticotectal fibers (the occipital and preoccipital divisions of the internal corticotectal tract of Crosby and Hender-

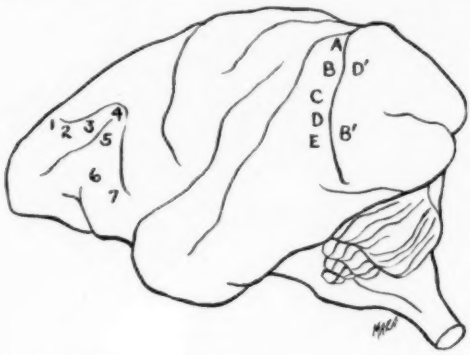

Fig. 2.—Left hemisphere of Macaca mulatta. Letters *A* through *E* indicate the localization pattern of conjugate movements on the preoccipital eye field (Area 19), as follows: *A* indicates deviation upward; *B*, obliquely upward to the right; *C*, to right; *D*, obliquely downward to right; E, downward. *D'* and *B'* represent conjugate movements from the occipital eye field (Area 18) as follows: *D'*, eyes obliquely downward to right; *B'*, eyes obliquely upward to right.

Numbers 1 through 7 represent localization areas on the frontal eye field as follows: 1 and 7, conjugate ocular deviation obliquely downward to right; 2 and 6, divergence; 3 and 5, conjugate right horizontal deviation; 4, conjugate deviation upward to right.

Note duplication of pattern on the frontal eye field.

son [7]) were traced to the superior colliculi (an undetermined amount of the tracts decussating in the superior collicular commissure). The bundles of this corticotectal system from the lower occipital and upper preoccipital fields terminated on rostral and medial portions of the superior colliculi; those fascicles from the upper portions of the occipital and the lower parts of the preoccipital field ended in the caudolateral regions of the superior colliculi. This pattern on the superior colliculi has been documented by Peterson and Henneman.[21] From the part of the preoccipital eye field (Area 19; Fig. 2, *C*) which produces horizontal conjugate devi-

21. Peterson, E. W., and Henneman, E.: Cortico-Collicular Connections in the Macaque, Tr. Am. Neurol. A. **73**:119-122, 1948.

ation of the eyes toward the side opposite stimulation, a corticotegmental system (Crosby and Henderson [7]) was traced into the midbrain and, by-passing the superior colliculi, was believed to descend, with some synapse in course, to the contralateral abducens nucleus.

We believe the localization patterns in the frontal eye fields of the macaque to be those illustrated in Figure 2 (and listed in the description of this figure). The most striking features are probably the duplication, in inverse order, of the pattern on the upper half of the field by that on the lower half, and the relatively large proportion of the whole field related to conjugate deviation of the eyes in the horizontal plane. Paths were traced from such areas devoted to horizontal deviation to the contralateral abducens nucleus (Crosby [17]; the abducens component of the corticobulbar tract of Crosby, Yoss, and Henderson [20]) and from the portion of the field yielding obliquely upward movements to the oculomotor nucleus. Simultaneous and equal bilateral stimulation of like appropriate areas yielded directly upward, or directly downward, deviation, or gaze straight ahead.

The modification of the patterns normally elicitable from the preoccipital and the occipital fields, with resulting increase, decrease, or absence of optokinetic nystagmus, is a matter of some interest. Such modification of the horizontal pattern will now be reported.

EXPERIMENTAL RESULTS

The results obtained from the seven monkeys tested during this study of optokinetic nystagmus are presented, in detail, in the following protocols.

MONKEY 7.—*Macaca mulatta; 3 to 4 lb. (1.3 to 1.8 kg.); female.*

Tests on June 28, 1949, showed fairly active, but somewhat irregular, optokinetic nystagmus. The responses were equal with rotation of the drum to either side. On July 5, the "horizontal" portion of the left preoccipital cortex (Area 19) was ablated, but the occipital eye field (Area 18) was spared (Fig. 3). No change in optokinetic response was noted immediately after the animal regained consciousness. It was felt that the tests were made too soon after the operation for conclusive results. On July 12, a partial ablation of the "horizontal" area of the left frontal eye field was performed (Fig. 3). On July 19, the optokinetic nystagmus was found to be greater with rotation of the drum away from the side of the frontal lesion than toward it. When the drum was rotated toward the side of this lesion, there was much less response, and the eyes tended to be held for short periods in the "eyes-right" position, with much less difference between the quick and the slow component. The animal died of a gastrointestinal infection on July 23, 1949.

MONKEY 9.—*Macaca mulatta; 3 to 4 lb.; female.*

On Sept. 27, 1949, the optokinetic responses were poorly elicited. The nystagmus was inhibited almost completely by voluntary fixation when the monkey was attentive to the world around her. As this attention to outside affairs lessened, her eyes presumably became fixed on the stripes of the drum, and a much better following movement occurred in the expected direction, with no distinct quick component of return. There was, however, some slight difference between the components. Admixtures of vertical movement occurred, directly related to efforts to maintain voluntary gaze, which broke up the horizontal response. With loss of attention, these vertical admixtures became less prominent. The animal died of hemorrhage at operation on Oct. 4, 1949.

MONKEY 11.—*Macaca mulatta; 6 to 7 lb. (2.7 to 3.2 kg.); male.*

On Oct. 4, 1949, no sustained optokinetic nystagmus could be produced. An occasional movement occurred in the proper direction, usually as attention had lagged. When the monkey was tense and watchful of the world beyond the drum, there was no optokinetic movement whatever. Less response was obtained than in Monkey 7, but Monkey 11 was older and larger. On Oct.

6, the right Area 8 was destroyed in part (Fig. 3). On Oct. 18, the monkey showed optokinetic nystagmus on rotation of the drum toward the side of the lesion and none on rotation in the opposite direction. This monkey was then utilized for other experiments.

MONKEY 14.—*Macaca mulatta; 4 to 5 lb. (1.8 to 2.3 kg.); sex not noted.*

On Jan. 24, 1950, with the animal under light ether anesthesia, the optokinetic nystagmus was perfect with respect to following and return components and with regard to the direction of movement of the drum. As the anesthesia became lighter, the nystagmus decreased in amount, in amplitude, and in specificity of direction. After a 20-minute waiting period, with the animal obviously fully awake, the nystagmus was much poorer and required a longer duration of

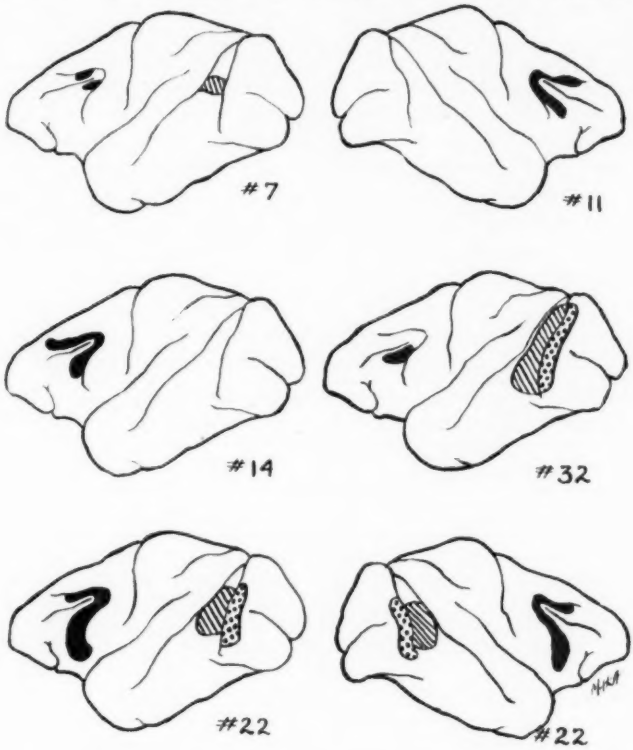

Fig. 3.—Diagrams of lesions described in the protocols of the similarly numbered monkeys. Lesions of the frontal eye field are indicated in solid black; lesions of the preoccipital eye field (Area 19) are shown by diagonal lines; lesions of the occipital eye field (Area 18) are indicated by black dots.

stimulation to be elicited. When it did occur, it was easily and frequently inhibited by voluntary fixation movements. The amplitude obviously varied with attention on matters other than the stripes of the drum; the difference between the following movement and the quick return was less marked, and at times the two movements were indistinguishable. As attention to affairs beyond the drum lagged, several typical beats of nystagmus occurred. The "wide-awake" response was directly comparable to that of the monkeys previously tested. On Jan. 31, the "horizontal" part of the left frontal eye field was localized by stimulation and destroyed (Fig. 3). On Feb. 7, no conclusive nystagmus was obtained, but with the drum rotated to the right

(that is, away from the side of the lesion) the eyes tended to jerk to the left and to stay there. The animal was then utilized, on Feb. 9, for a study of the localization pattern in the frontal eye field.

MONKEY 19.—*Macaca mulatta; 3 to 4 lb.; female.*

On July 27, 1950, a typical optokinetic response was obtained with the animal under light ether anesthesia. With partial recovery from the anesthesia the eye movements became pendular with the more marked movement away from the direction of rotation of the drum. With a return of consciousness almost complete inhibition of the following movements occurred. This animal was then used, on Aug. 3, for stimulation of the left frontal eye field and for placement of destructive lesions. There was no further optokinetic testing.

MONKEY 22.—*Macaca mulatta; 3 to 4 lb.; female.*

On Sept. 21, 1950, with the animal awake, optokinetic nystagmus, pendular and erratic, was present to either side. These responses improved when the animal was drowsy or inattentive to the world around her, and so focused her eyes automatically on the stripes on the drum. On Oct. 4, large bilateral lesions were placed in the frontal eye field (Fig. 3). On Oct. 19, the optokinetic responses were more marked than those obtained at any time in any monkey tested at full consciousness. They were typical, persistent, the same in either direction, and continued for at least 15 seconds after the drum was stopped. Changes in the direction of gaze did not alter the responses. On Oct. 25, bilateral cautery lesions were made in Area 19 (Fig. 3). Testing on Oct. 26 showed aimless eye movements, with poor following movements. The optokinetic response was poor, but occasional unsustained movements were obtained in the proper direction on rotation of the drum. With continued stimulation, responses disappeared entirely. The type of nystagmus produced was reduced in amount, was of smaller amplitude, and had a less specific direction than that before any operation had been performed. On Oct. 30, the occipital eye field (Area 18) was removed bilaterally (Fig. 3). The monkey was tested for optokinetic nystagmus on Nov. 2. No response was obtained on testing by rotation of the optokinetic drum. No following movements could be elicited in other ways. The animal was killed for microscopic study on Nov. 9, 1950.

MONKEY 32.—*Macaca mulatta; 5½ lb. (2.5 kg.); male.*

On Feb. 1, 1951, the optokinetic nystagmus was poor and unsustained. When present, the movements occurred in the expected direction, but there was a definite tendency to hold the eyes laterally in the direction of the expected quick component. Obviously, there was a marked inhibition or suppression of the optokinetic response. On Feb. 5, the preoccipital and occipital eye fields were removed on the left (Fig. 3). Tests on Feb. 8 showed that the only response was a tendency to hold the eyes in the right conjugate position when the drum was rotated toward the side of the lesion. No optokinetic response was obtained when the drum was rotated away from the side of the lesion. On Feb. 15, the ventral subfield of the left frontal eye field was cauterized (Fig. 2, 5). On Feb. 22, optokinetic nystagmus was present and sustained on rotation of the drum toward the side of the lesions. It was equivocal toward the other side. The animal was killed on March 3, 1951.

CORRELATION OF RESULTS

In the normal monkey the nystagmus set up by rotation of the optokinetic drum is modified frequently as to amplitude or direction (Monkeys 14 and 22), is unsustained (Monkey 32), or may be suppressed (Monkeys 9 and 11) by voluntary fixation (probably by looking beyond or through the drum). When optokinetic nystagmus does occur, its slow component is toward the side of the expected deviation of the eyes (Monkey 7), that is, in the direction of rotation of the drum, for the slow component is produced, in the intact animal, by a continuous excitation of the following movements which constitute an automatic response to prolonged visual stimuli. Too deep an anesthesia will abolish optokinetic responses, but these may reappear as the effects of the anesthetic agent gradually wear off.

Complete bilateral destruction of the portions of the preoccipital field (Area 19) concerned functionally with conjugate horizontal deviation of the eyes and of the bordering zone of the occipital field (Area 18, and possibly some of Area 17 in the macaque), of supposedly like significance, abolishes optokinetic responses (Monkey 22). It may be concluded, then, that such preoccipital and occipital fields are necessary parts of the pathways involved in optokinetic responses in the horizontal plane.

Destruction of the preoccipital eye field, either unilaterally (Monkey 7) or bilaterally (Monkey 22), does not prevent the optokinetic responses away from the side of the lesion, although after such destruction they may be reduced in amount and in amplitude and may be less specific in direction (Monkey 22).

If only the portions of the preoccipital area (Area 19) related to horizontal deviation of the eyes and the bordering zone of the occipital eye field (Area 18) are removed unilaterally (Monkey 32), no definite optokinetic responses are obtainable when the drum is rotated away from the side of the lesion. When, in such a unilateral destruction, the drum is rotated toward the side of the lesion, if the animal's attention lags, a true optokinetic response may result, with the following component of the nystagmus directed away from the uninjured half of the brain. However, if the animal is watchful, voluntary inhibition of the latter response may occur, and then the eyes may not respond to the rotation of the optokinetic drum or may even be held toward the uninjured side in overcompensation (Monkey 32). If, then, any portion of the frontal eye field related to horizontal deviation of the eyes is destroyed on the same side as the lesion in the preoccipital and occipital fields (Monkey 32), it is obvious that any inhibitory or suppressor effect that this part of the frontal eye field may have over the contralateral, uninjured preoccipital and occipital fields will be lost. To test the possibility of such an inhibitory function, in Monkey 32 the ventral subfield of the frontal eye field (Fig. 3) was destroyed on the same side as the preoccipital and occipital lesions. Tests with the optokinetic drum then showed consistently, even though the monkey was wide-awake, a fairly good, sustained optokinetic nystagmus when the drum was rotated toward the side of the lesions, whereas before the last operation the monkey was able to suppress such a nystagmus. No optokinetic response was obtained, of course, when the drum was rotated away from the side of the injured preoccipital and occipital fields. The results just discussed are regarded as providing evidence of an inhibitory effect of certain portions of the frontal eye field over the contralateral occipital and preoccipital eye fields.

With bilateral destruction of the frontal eye fields, but with preoccipital and occipital fields undisturbed (Monkey 22), heightened and sustained optokinetic responses were obtained in either direction on testing with the optokinetic drum. Such responses were more marked than those elicited in any of the other experiments. The eye movements were typical and the same in both directions, and they tended to persist for some time after the cessation of the stimulation. Animals under light anesthesia, during which the frontal lobe regions are presumably depressed, show also a marked increase in optokinetic responses (Monkeys 14 and 19). Such results afford an excellent illustration of the character of unopposed optokinetic responses and also of the strongly inhibitory (or dominant) influence of the frontal eye fields over appropriate parts of the preoccipital and occipital eye fields in the macaque.

The idea that the frontal eye fields may exert inhibitory, as well as excitatory, effects over other parts of the nervous system has been expressed by various observers. Sherrington [22] demonstrated that each frontal eye field has a dual functional relation to the lower motor centers innervating eye muscles. Thus, its discharges not only activate the neurons which innervate the muscles concerned in turning the eyes in a given direction, but lessen or inhibit the flow of impulses through those nerve cells which innervate the antagonistic muscles. Russell [23] determined the strength of stimulus necessary to turn the eyes toward the right by excitation of the left frontal cortex and that required to produce horizontal conjugate deviation of the eyes to the left on stimulation of the right occipital area, which, in turn, would set off a discharge from the appropriate portion of the neighboring preoccipital field. Then, utilizing these data, he stimulated the appropriate regions of the left frontal and the right occipital eye field simultaneously and obtained conjugate horizontal deviation of the animal's eyes toward the right, the characteristic response for stimulation of the left frontal eye field. Such an experiment certainly indicates that the discharge of the frontal eye field is dominant over that of occipital and preoccipital eye fields. McCulloch's [19] studies have demonstrated that excitation of Area 8 (which falls within the frontal eye field) may result in suppression of the homolateral and contralateral Area 19 (which includes the preoccipital eye field) and facilitation of the contralateral and homolateral Area 18. Inhibition or suppression is usually dominant over facilitation. The interrelations of Areas 18 and 19 are such as to further eye movements in like direction (see earlier discussion on page 5). Consequently, a unilateral lesion placed in the "horizontal" portion of one frontal eye field (Fig. 2), in the absence of other brain lesions, will not interfere seriously with voluntary inhibition of optokinetic responses toward either side. After such a unilateral ablation, if the monkey's attention is not fixed on the drum (even though he is looking directly toward it) but, instead, because of fear or curiosity, is centered on the world beyond the drum, he may suppress optokinetic responses toward either side (Monkey 11, away from the side of the lesion) or toward both sides (Monkey 14). On the contrary, if his attention is fixed on the drum, he may exhibit optokinetic nystagmus to either side (Monkey 11, toward the side of the lesion) or to both sides (Monkey 7), but greater away from the side of the lesion.

Certain of the results obtainable on testing Monkeys 7 and 32 with the optokinetic drum may appear inconsistent, but such inconsistency is apparent, not real. The results obtained with these monkeys, too, depend on the complicated interrelations of the various portions of the cerebral cortex related to ocular deviations. It is obvious, as has already been pointed out, that ablation of the horizontal portion of one preoccipital field only will not prevent optokinetic responses away from the side of the lesion (Monkey 7), but that unilateral destruction of the portions of the preoccipital and occipital fields related to horizontal following movements (Monkey 32) will do so. Destruction of a preoccipital field only may actually further optokinetic responses away from the side of the lesion, since neither frontal eye field can then

22. Sherrington, C. S.: Further Experimental Note on the Correlation of Action of Antagonistic Muscles, Proc. Roy. Soc. London **53**:407-420, 1893.

23. Russell, J. A. R.: An Experimental Investigation of Eye Movements, J. Physiol. **17**:1-26, 1894.

inhibit such discharges. The horizontal portions of the frontal eye fields—even that of one frontal eye field (as in Monkey 7 after operation)—may still suppress optokinetic responses toward the side of the lesion if the monkey's attention is centered on the world beyond the drum, although such responses may occur if he attends to the drum. There is a growing body of evidence (Crosby, Yoss, and Henderson [20]; Crosby [24]) to suggest that the upper and lower parts of the horizontal portion of each frontal eye field (Fig. 2) have different functions, but this evidence is not ready for presentation as yet. It is probable, however, that a partial lesion in the horizontal portion of one frontal eye field may reduce its suppressor effect over the preoccipital eye fields more than will a total ablation of the horizontal portion of such a frontal field. This is believed to be the case, at least, when, as in Monkey 32, the lower part of such a horizontal portion of one frontal eye field is ablated and the upper portion is spared. This monkey showed marked optokinetic responses when the drum was rotated toward the side of the lesion.

The results presented here do not agree in some particulars with the conclusions reached by Cogan and Loeb [3] in their recent study of optokinetic nystagmus in patients with unilateral lesions of the cerebral hemisphere. The explanation for these differences may lie in the fact that the authors were dealing with lesions involving very considerable areas of the brain; we, with small isolated lesions. Other regions of the cortex beside the frontal eye fields discharge to the preoccipital areas. Or, it is possible that the association pattern is different or the frontal lobe dominance less in man than in the macaque.

SUMMARY OF EXPERIMENTAL RESULTS WITH MONKEY

1. Intact preoccipital (Area 19) and occipital (Area 18) eye fields are essential for normal optokinetic responses. If both these fields are ablated bilaterally, such responses are abolished.

2. If the appropriate portions of the preoccipital and the occipital eye fields are destroyed unilaterally, optokinetic responses are not elicited on rotation of the drum away from the side of the injury.

3. If the portion of one preoccipital eye field functionally related to conjugate horizontal deviation of the eyes is destroyed, the homolateral occipital eye field may still produce an optokinetic response in the expected direction.

4. Each frontal eye field appears to exert an inhibitory or suppressor effect over the activity of the contralateral and homolateral preoccipital eye fields with respect to optokinetic responses in the horizontal plane. Unilateral ablation of a frontal eye field still permits regulation of the optokinetic responses in either direction by the intact frontal field. This fact may explain some of the apparent reversals in optokinetic responses reported in clinical practice.

5. Bilateral destruction of considerable portions of the frontal eye fields, including those regions related to horizontal deviation of the eyes, makes possible maximal optokinetic responses in the horizontal, and probably in other, planes.

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24. Crosby, E. C.: Central Nervous System Control of Eye Movements, 35th Mellon Lecture, University of Pittsburgh School of Medicine, Nov. 6, 1951, to be published.

CLINICAL AND PATHOLOGICAL CHARACTERISTICS OF RADIATION CATARACT

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RECENT interest in the effects of radiation on the eye in general and in the production of radiation cataract in particular makes it timely to inquire what the characteristic findings are in this type of cataract and how pathognomonic they are for radiation.

Accordingly, the clinical findings have been reviewed in 20 early cases of radiation cataract which we have seen, and the histologic changes have been studied in cross sections from 27 cases [1] of radiation cataract, representing various stages of cataract formation.

CLINICAL MANIFESTATIONS

The material comprising the clinical study consisted of 7 cases of cataract following exposure to x-rays or gamma-rays, 2 cases of cyclotron-induced cataract, 10 cases of cataract attributed to the ionizing radiations of the atomic bomb in Japan, and 1 case of cataract resulting from an atomic explosion in this country.

The findings were remarkably uniform in all cases.

On ophthalmoscopic examination the initial opacity appeared to consist of a dot, usually at the posterior pole of the lens, and as this increased to 1 to 2 mm. in diameter there appeared pepper-like granules and vacuoles scattered about it (Fig. 1). The main opacity then developed a relatively clear center, producing a doughnut-shaped (or occasionally multilocular) pattern with an over-all diameter of 3 to 4 mm. About this time granular opacities and vacuoles were found in the anterior subcapsular zone, predominantly in the pupillary area. These changes

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From the Howe Laboratory of Ophthalmology, Harvard University Medical School, and the Massachusetts Eye and Ear Infirmary, and the Department of Ophthalmology, Columbia University.

1. The two specimens resulting from atomic-bomb radiation were contributed by Dr. H. Ikui; one specimen of a radiation cataract was contributed by Dr. Wilfred Frye, and one, by Dr. Ed Maumenee and Dr. Frank Winter.

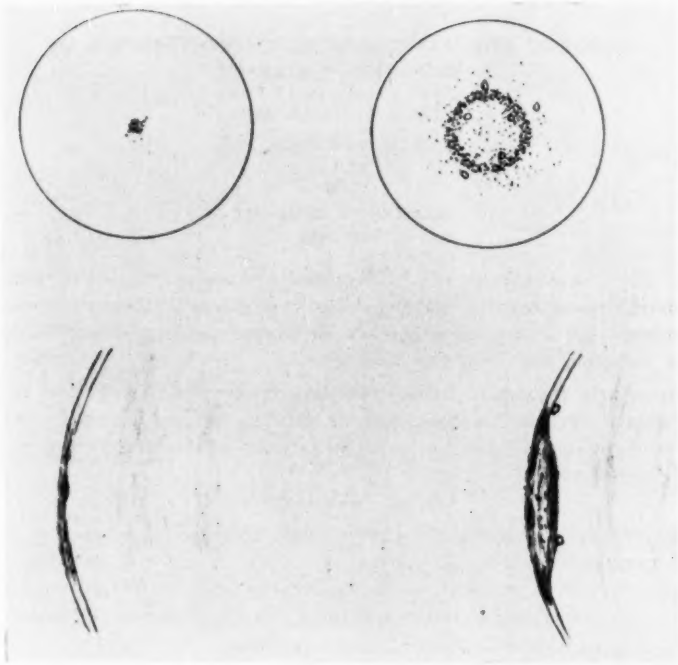

Fig. 1.—Diagrammatic representation of early clinical appearances in radiation cataract. The drawing in the upper left corner represents the opacity at the posterior pole of the lens as seen with the ophthalmoscope, and that in the lower left corner, as it is seen with the slit-lamp beam. Noteworthy is the apparent limitation of the early opacity to the plane of the posterior capsule. The drawings in the upper and lower right corners show the ophthalmoscopic and slit-lamp appearances at a later stage, forming the characteristic doughnut silhouette and bivalve configuration of the opacity.

appeared to be stationary or were only slowly progressive over a period of several years. Later, progressive opacification of the cortex occurred, forming eventually a mature and nonspecific cataract. The ophthalmoscopic appearance of radiation cataract has been pictured diagrammatically by Leinfelder and Kerr [2] and by Kimura and Ikui.[3]

With slit-lamp biomicroscopy, the initial opacities appeared granular and usually yellow. One of us believed that a golden-yellow reflex about the posterior capsule could be appreciated before any real opacification was apparent. In the early stages of cataract formation the opacities were in the plane of the posterior capsule. They were amorphic, frequently accompanied with polychromatic crystals, and have been likened in their distribution to "wind tracks on sand." The vacuoles, when present, were always situated just anterior to the plane of these opacities.

At a later stage the opacities split parallel with the plane of the capsule to form a bivalve configuration. This is the biomicroscopic counterpart of the doughnut silhouette seen ophthalmoscopically, with the central clear area corresponding to the split in the opacity and the peripheral opaque ring corresponding to the fusion of the anterior and posterior laminae. While the posterior lamina of opacity remained continuous with that of the posterior capsule, the anterior lamina changed progressively from a concave-forward to a convex-forward line. Especially noteworthy, however, was the fact that this line was always smooth on its anterior face and sharply demarcated from the rest of the lens. Only late did the flocculent cortical opacities develop; these may be differentiated from the opacities described above in that they are white, have jagged edges, and are situated anterior to the posterior capsule.

These changes, with minor variations, were seen in all the cases studied and are in accord with descriptions reported by others except that the limitation of the initial opacity to the posterior capsular region has not been noted. Previously it had been generally held that the initial opacity was in the cortex. The bivalve pattern and doughnut appearance were especially well illustrated by Rohrschneider [4] and by Vogt.[5] Meesman,[6] and later Grzedzielski,[7] pointed out the characteristically sharp demarcation of the opacity from the rest of the cortex.

The two types of cataract which may be most easily confused with the radiation type are posterior polar cataracts, such as are seen with retinitis pigmentosa, and the so-called cataracta complicata, such as occurs with endogenous ocular and systemic diseases and often without apparent cause. Both these types are unlike radiation

2. Leinfelder, P. J., and Kerr, H. D.: Roentgen-Ray Cataract: An Experimental, Clinical, and Microscopic Study, Am. J. Ophth. **19**:739-756, 1936.

3. Kimura, S. J., and Ikui, H.: Atomic-Bomb Radiation Cataract: Case Report with Histopathologic Study, Am. J. Ophth. **34**:811-816, 1951.

4. Rohrschneider, W.: Klinischer Beitrag zur Entstehung und Morphologie der Röntgenstrahlenkatarakt, Klin. Monatsbl. Augenh. **81**:254-259, 1928.

5. Vogt, A.: Der Röntgen- und Radiumstar, in Vogt, A.: Lehrbuch und Atlas der Spaltlampenmikroskopie der lebenden Auges, Berlin, Julius Springer, Vol. II, p. 709-714.

6. Meesman, A.: Beitrag zur Röntgen-Radiumstrahlenschädigung der menschlichen Linse, Klin. Monatsbl. Augenh. **81**:259-269, 1928.

7. Grzedzielski, J.: Zur Histologie der Röntgenkatarakt, Klin. Monatsbl. Augenh. **95**:360-369, 1935.

cataract in having at the outset flocculent cortical opacities extending a millimeter or more into the cortex in front of the posterior capsule. As the cataracts mature, the findings become identical irrespective of the etiologic type; hence the distinguishing features are valid only in the relatively early stages of cataract formation. The lenticular changes in the one patient with glassblowers' cataract seen by us were identical with those caused by ionizing radiation.

While these generalizations hold sufficiently well to be useful, they are not without exceptions. Thus, an opacity that appeared to be in the posterior capsule, and even had the bivalve configuration, was seen in the lens of a patient not known to have had any significant radiation. The irides in this patient were tremulous, and there were perinuclear rider-like opacities, suggesting a congenital abnormality. The possibility that the patient's mother had been irradiated during pregnancy could not be excluded. Another patient with presumed cataracta complicata accompanying

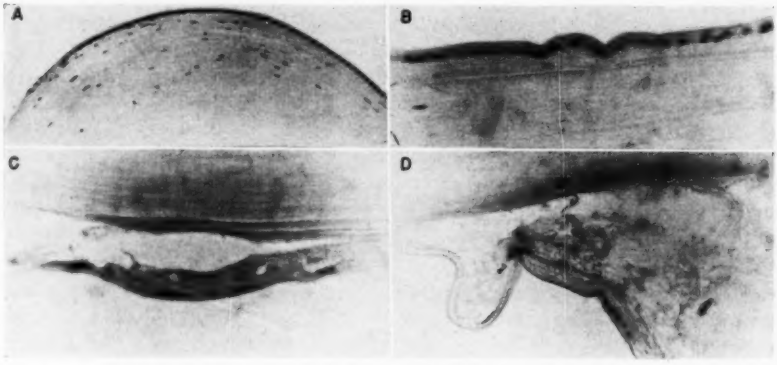

Fig. 2.—Specimens of a lens which had received 8,850 r of x-radiation (198 kv.) two years previously, given over a four-month period for retinoblastoma. At the time of enucleation the patient was 3½ years old and showed an incipient cataract. The photographs are intended to illustrate early changes in x-ray cataract: *A*, an essentially normal equator except for loss of the bow configuration of nuclei and extension of some cells beneath the posterior capsule; *B*, normal anterior lens epithelium except for an occasional hiatus, corresponding to loss of a single epithelial cell; *C*, posterior region of the lens, showing a few cells immediately beneath the capsule and a small amount of subcapsular debris; *D*, another part of the posterior lens, showing a few cells beneath the capsule and fibrillary thickening of the posterior capsule.

rheumatoid arthritis showed in one eye a shell-like and yellow opacity of the posterior capsule, similar to that in radiation cataract, but only cortical opacities in the other eye.

HISTOPATHOLOGIC CHARACTERISTICS

Reports in the literature on the histology of radiation cataract in human beings have been remarkably few. Grzedzielski [7] described proliferation of cells in the posterior subcapsular region and suggested, contrary to the views of Rohrschneider and Vogt, that the opacities seen clinically were due to disintegration of these cells rather than to a direct effect of the radiation on the lens substance. Leinfelder and Kerr [2] described swelling of lens fibers at the equator and degenerating cells beneath the posterior and anterior capsules but did not believe that their evidence indicated

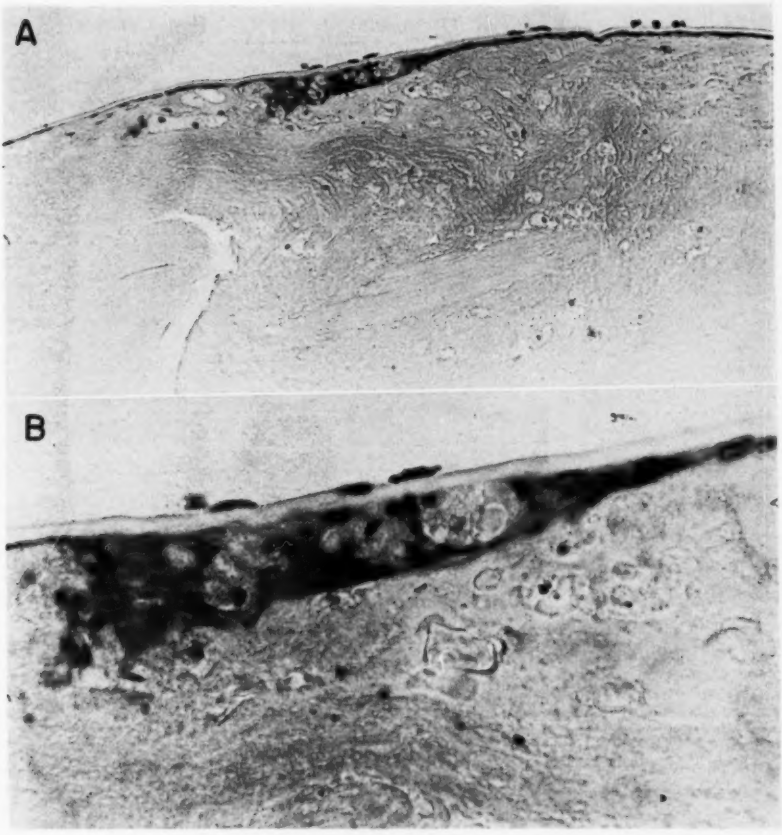

Fig. 3.—Equatorial region of lens from an eye which had received 3,500 r (140 kv.) of x-radiation several years previously for an epithelioma of the lid. This resulted in a cataract, which was fairly advanced at the time of extraction. The photographs (*A*, low power; *B*, high power) show a piling-up of cells at the equator with extension of cells posteriorward and lack of differentiation of the cells into lens fibers.

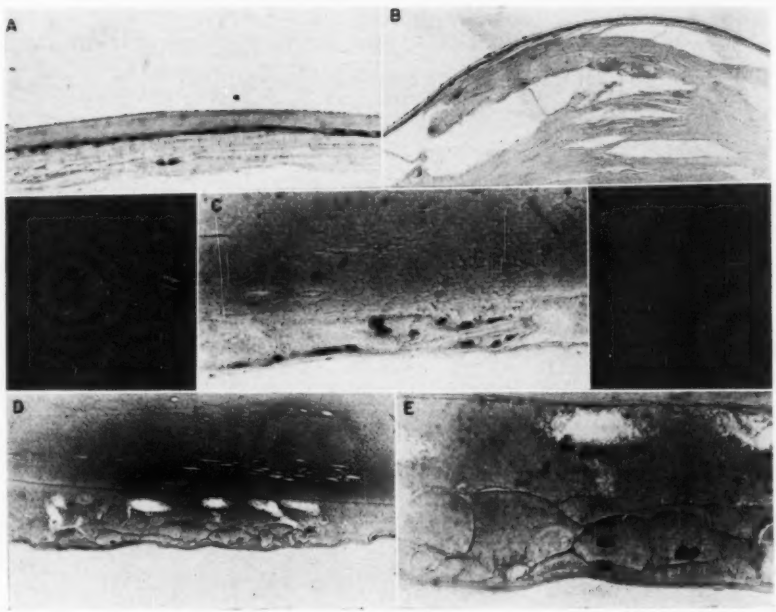

Fig. 4.—Portions of lens from an eye which had been exposed to radiation during treatment for an epithelioma of the lower lid four years previously, with subsequent development of a radiation cataract. The age of the patient was 58. *A*, considerable attenuation of cells of the anterior-capsule epithelium with numerous hiatuses, corresponding to loss of cells and an occasional "dropping-off" of the cells into the anterior cortex; *B*, lack of differentiation of cells in the cortex of the equatorial region and extension of epithelium posteriorward; *C*, cells beneath the posterior capsule with configuration suggesting abortive differentiation into lens fibers; *D* and *E*, "balloon-cell" formation beneath the posterior capsule, representing a metamorphosis of cells from a spindle-shaped structure immediately beneath the capsule to a swollen and occasionally anuclear, but well-defined, mass farther inward, with the ultimate formation of amorphous debris containing a few free-floating nuclei.

a proliferation of cells, such as Grzedzielski had described. Reese [8] emphasized the metaplasia of the anterior-lens epithelium in situ and pointed out that this, together with the cortical liquefaction, accounted for the anterior subcapsular haze seen clinically. He also described abnormalities in the epithelial cells at the equator and migration of cells beneath the capsule posteriorly. Krückmann [9] also noted the presence of epithelial cells lining the posterior capsule in the case of an x-ray cataract which he studied, but his was a relatively mature cataract, and such a migration of cells posteriorward is a common finding in the late stage of many types of cataract. Hence it was impossible for him to infer which changes were attributable to radiation.

Kimura and Ikui [3] described in detail the clinical and histologic changes in the radiation cataract of one of the atomic-bomb survivors. The chief abnormalities noted were irregularity in cells at the equator and the presence of an amorphous material between the capsule and the remaining cortex. This material was thought to result from corticolysis rather than to be derived directly from the epithelial cells.

The specimens obtained for the present study consisted of 24 cases in which the cataract resulted from therapeutic radiation for tumor in or about the eye, 2 cases in which the cataract had resulted from the ionizing effect of the atomic bomb, including the case reported by Kimura and Ikui,[3] and 1 case of cataract caused by cyclotron exposure. Of these, the cataractous changes may be said to have been in an immature stage in seven cases.

The lesions found in those cataracts which were least advanced consisted of the following: attenuation and spottiness of the anterior subcapsular epithelium (Figs. 2*B* and 4*A*); piling up of equatorial cells, with failure of the cells to "drop off" into the cortex (Figs. 3*A* and *B*; 4*B*; 6*A*); presence of cells with spindle-shaped nuclei beneath the posterior capsule (Figs. 2*C* and *D*; 3*A*; 4*C* and *D*; 6*D*; 7*B*); balloon-cell formation at the equator and posterior subcapsular region (Figs. 4*D* and *E*; 5*A* and *B*; 6*E*); presence of a few phagocytes on the outside of the capsule (Figs. 7*A* and *B*), and amorphous debris in the region of the posterior cortex. This debris, which has heretofore been unequivocally accepted as liquefied cortex, predominated in the posterior subcapsular region and was sharply demarcated from the hyaline cortex anterior to it. With advanced cataracts, there were apparent lysis of the cortex, extension of epithelium posteriorly, loss of differentiation at the equator, and, finally, complete disappearance of all epithelium.

None of these changes can be considered pathognomonic of radiation cataract, for they are found to varying degrees in other types of cataract (e. g., Fig. 8*A* and *B*). The observations in three cases, however, are especially noteworthy in presenting unusual findings which do not appear to have been reported in other types of cataracts. All three lenses showed a fibrillary thickening of part of the posterior capsule. Serial sections were made in two of these cases.

In the first of these three cases a lens was removed intracapsularly by Dr. H. Ikui from a Japanese man aged 25 who had been within 1 km. of the hypocenter at

8. Reese, A. B.: Operative Treatment of Radiation Cataract, Arch. Ophth. **21**:476-485 (March) 1939.

9. Krückmann, J.: Über den histologischen Befund einer Röntgenkatarakt, Klin. Monatsbl. Augenh. **104**:621-628, 1940.

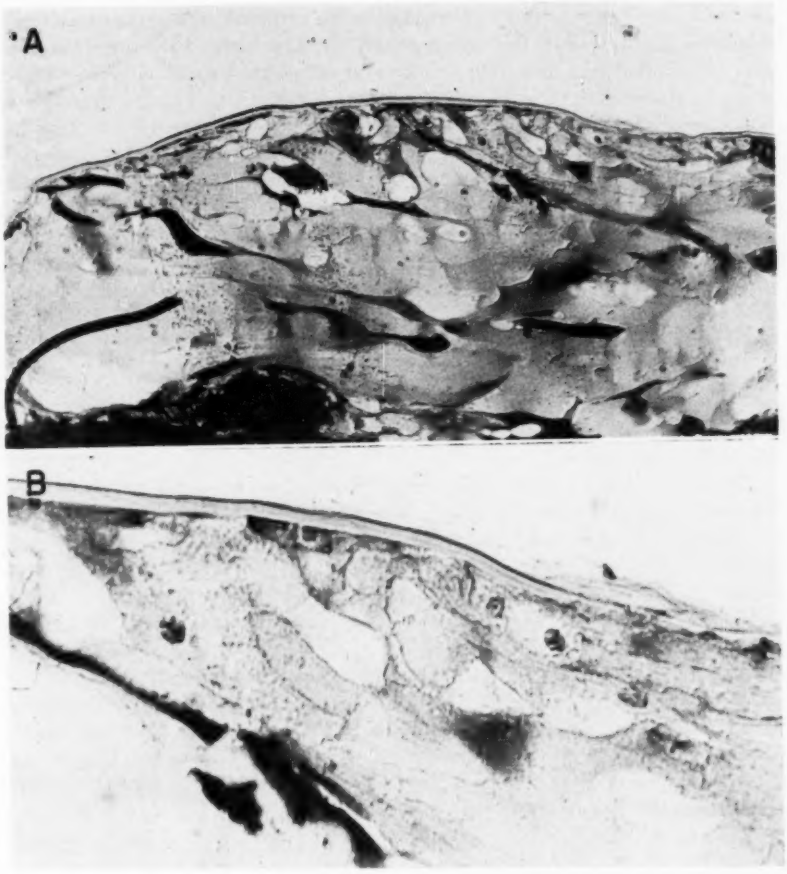

Fig. 5.—Posterior portions of a lens stained by the Masson trichrome technique from a rabbit eye which had received 1,500 r (200 kv.) of x-radiation 150 days prior to enucleation. At the time of enucleation the lens was entirely opaque. The specimens (*A*, low power; *B*, high power) show the same metamorphosis as that indicated in the specimens shown in Figure 4, consisting of a transition from spindle-shaped cells adjacent to the posterior capsule through a stage of nucleated "balloon cells" to non-nucleated "balloon cells," and ending finally in amorphous debris in the subadjacent cortex.

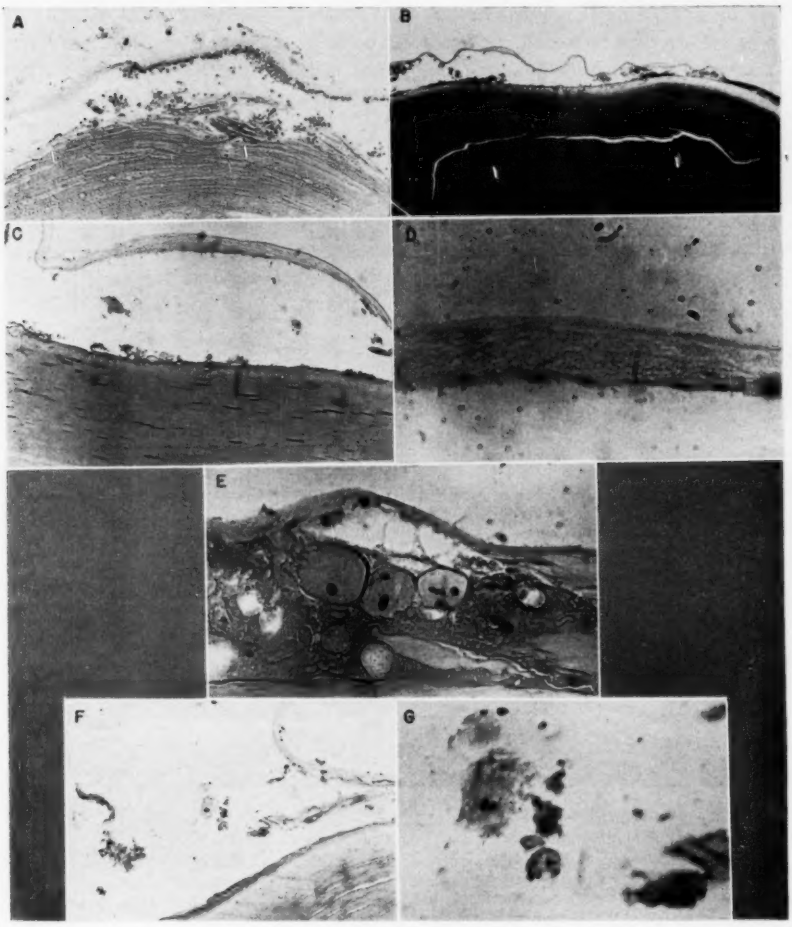

Fig. 6.—Lens from a Japanese man aged 25 who was within 1 km. of the hypocenter at the time of the Hiroshima explosion and in whom a radiation type of cataract developed approximately 2½ years later. At the time of the extraction the lens showed an opacity which appeared to be within the posterior capsule and showed the characteristic bivalve splitting with slit-lamp biomicroscopy and the doughnut-shaped opacity with ophthalmoscopy.

The areas photographed are intended to illustrate the following changes: *A*, piling-up of cells at the equator and failure of cells to differentiate into lens fibers; *B*, double area of thickening of the capsule in paraxial areas with disorganized material between capsule and cortex; *C*, higher magnification of one of the thickened areas, showing the fibrillary nature of the thickening and the fact that the thickening remains with the capsule when the latter is separated from the cortex; *D*, still higher magnification, showing that spindle-shaped cells are affixed to the posterior surface of the thickened area, as to the capsule elsewhere; *E*, "balloon-cell" formation beneath the posterior capsule, similar to that which has been described in the preceding cases of radiation cataract; *F* and *G*, areas adjacent to the place where the capsule had ruptured during the process of fixation, showing the cellular nature of some of the subcapsular debris. Especially noteworthy is the presence of a binucleate cell, whose cell wall appears to have ruptured and is presumed to be on the verge of forming the amorphous cytoplasmic debris and free-floating nuclei that make up much of the subcapsular material.

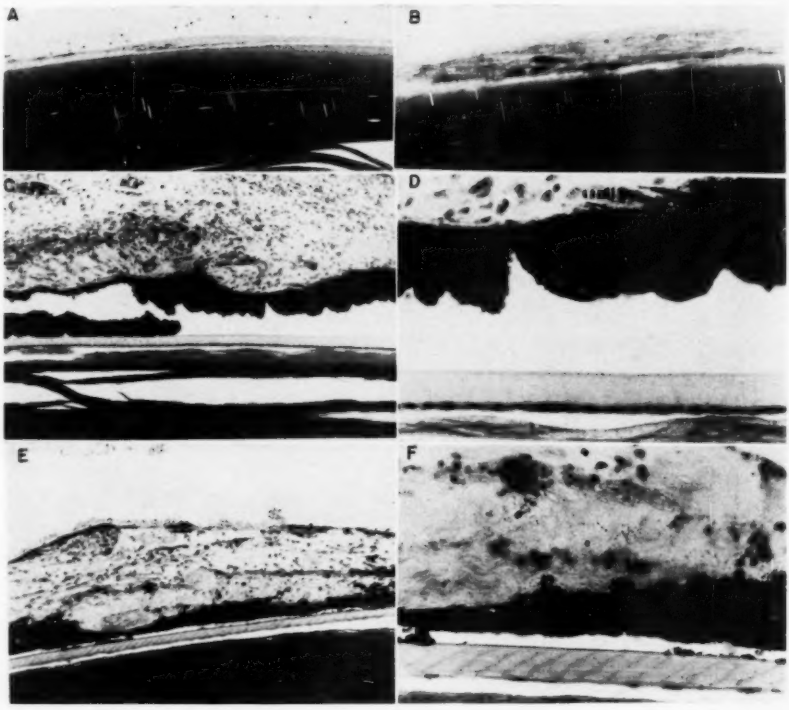

Fig. 7.—Specimens from an eye which had been treated with radium for a carcinoma of the limbus and which at the time of enucleation had an immature cataract.

A and *B*, low- and high-power magnifications, respectively, of one of the paraxial areas of capsular thickening. As in the previous case, the thickened area was continuous with the capsule and had the same staining characteristics. Evident in the present photographs are the apparent entrapment of nuclei within the fibrillary meshes of the thickened area and the presence of a few phagocytes outside the capsule.

C and *D* and *E* and *F* represent low- and high-power views from corresponding areas on either side of the pupil. The areas in the former (*C* and *D*) are interpreted as being entirely normal except for the artifactual adherence of the pigment epithelium to the capsule. The latter (*E* and *F*) show almost complete disappearance of the lens epithelium locally and necrosis of the posterior layers of the iris. In addition to the disappearance of the nuclei in the sphincter region, shown in the photographs, bleached specimens revealed almost complete disappearance of the nuclei in the pigment epithelium in a corresponding area.

the time of the Hiroshima explosion. Two to three weeks later the patient had complete, although temporary, epilation of the head region. The cataracts began to form approximately 2½ years after the explosion, and when the present lens was removed, five years after the explosion, there were a few vacuoles and opacities in the anterior cortex and an opacity of the posterior capsular and subcapsular region, forming the characteristic doughnut shape previously described. In the second case a cataract had developed in an eye after radium treatment of a squamous carcinoma

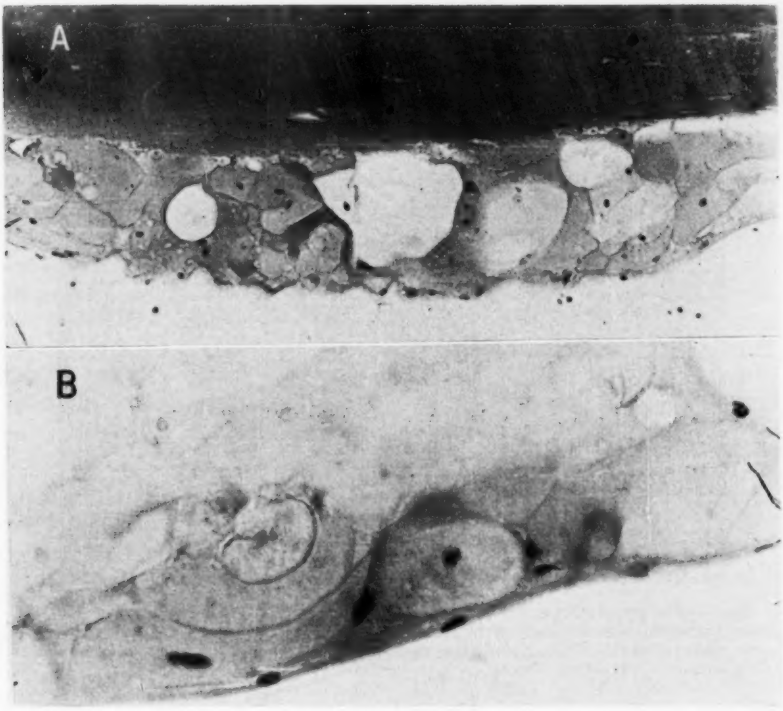

Fig. 8.—"Balloon-cell" formation in the posterior lens region of cataractous lenses not known to have received any radiation. *A* is from a mature cataract, presumably of the senile variety, and *B*, from the lens of a patient with choroideremia. Both show the progressive metamorphosis from spindle-shaped cells, in the subcapsular region, to amorphic debris, presumably resulting from ultimate rupture of the cells, in the deeper regions.

of the limbus. Information on the total amount of radiation was not available, and the clinical appearance of the cataract could not be determined because of corneal ulceration. Judging from the histologic changes, the cataract was in a relatively immature stage. In the third case the patient had received 3,000 r (1.2 mev) of x-radiation to the eye two years prior to enucleation. The clinical examination of the lens prior to enucleation was inconclusive because of corneal ulceration.

The histopathologic changes in these cases consisted of a fibrillary thickening of the posterior capsule to approximately three to four times normal (Figs. 2*D*; 6*B*, *C*, and *D*; 7*A* and *B*). In the first two cases, the areas of thickening extended over an area of 2 to 3 mm., and in some sections, presumably those through the posterior pole, there were two such areas, one on either side of the axis, leaving a central area of normal capsule 2 to 3 mm. in length. Serial sections in one case showed these two areas to converge and ultimately to fuse in one case, while in the other case they remained separate in all sections; it is noteworthy that the thickening might well have been overlooked had the sections not happened to be axial or immediately paraxial. In both cases the thickened areas were not seen as the sections

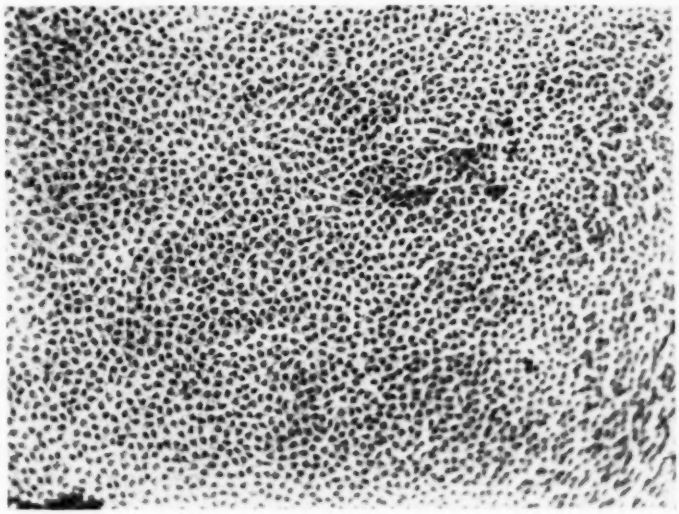

Fig. 9.—Flat preparation of lens epithelium of the rabbit. The extreme left edge of the photograph corresponds approximately to the anterior pole of the lens, while the right side corresponds to the equatorial region. The photograph is intended to show the "cracking" or separation of cells as the equator is approached (Zenker-fixed specimen).

became more peripheral. The distribution of the thickened areas was thought to correspond in general to the doughnut-shaped opacity that is seen clinically.

Although the thickened areas could be well seen in sections stained with hematoxylin and eosin, they were especially evident in sections stained with the Masson technique, in which the thickened zone took the same light-blue stain as the capsule and stood out in contrast to the dark red of the cortex and the subcapsular debris.

The thickening was characteristically fibrillar, and in this respect it differed from the uniform hyaline consistency of the normal capsule. The thickened fibers formed a meshwork continuous with the anterior portion of the capsule and had a variably defined anterior surface, often being faced with a lamina of cells. With artifactual separation of the capsule, the thickened area always remained with the capsule

(Fig. 6*C*). Entrapped within its mesh, cells could often be seen, giving rise to the impression that the thickening was in fact a product of the cells. (Fig. 7*A* and *B*).

In the third case there was also fibrillary thickening of the capsule, which was identical with that in the foregoing two cases and was also present in two zones on either side of the posterior pole. Serial sections, however, were not made.

A review of lenses showing cataracts not associated with radiation failed to reveal any similar thickening of the capsule, and sections of approximately 50 rabbit lenses, representing various stages of radiation cataract, failed to show any thickening of the capsule except in one mature cataract, in which there was a doubling of the capsule in a zone adjacent to the equator. This was interpreted as being of questionable significance.

It must be noted that Schlaegel [10] reported a thickening of the capsule in some of the lenses removed from atomic-bomb victims dying of radiation sickness within the first few weeks of the bomb explosion. These do not appear to have been the localized fibrillary variety here described, and there is no convincing evidence that they were associated with cataract.

In the case cited in which the patient had received radium radiation for an epibulbar carcinoma, a further observation may be noted as of interest. The iris showed localized necrosis (disappearance of nuclei with hyalinization of stroma and pigmented epithelium) in a 2 mm. zone on one side of the pupil and local disappearance of the underlying lens epithelium (Fig. 7*E* and *F*; compare with Fig. 7*C* and *D*). Elsewhere the epithelium appeared normal. Whether or not the findings in this case are related to radiation cannot be proved; the changes were not found in the other cases studied, but it is noteworthy that Leinfelder and Kerr described atrophy of the iris occurring two years after irradiation in one of their patients studied clinically, and one of us has made similar clinical observations.

No flat preparations of the lens capsule from radiation cataract in human eyes were available. However, flat preparations from approximately 20 senile cataracts were studied, and none of them showed the pleomorphic and multinucleate cells that have been found characteristic of radiation effects in rabbits.[11]

PATHOGENESIS OF RADIATION CATARACT

Before we attempt an interpretation of the clinical and histologic changes, it may be well to review the processes concerned with the normal maturation of lens fibers at the equator.

In passing from the center to the periphery, the first change in the lens epithelium seen in flat preparations is the development of fissures between the cells (Fig. 9). This is especially evident in Zenker-fixed tissue. It may be interpreted as a loosening of the individual cells prior to separation into the lens fibers. The rest of the changes, seen best in cross sections, consist of a "dropping-off" of the cells into the

10. Schlaegel, T. F.: Ocular Histopathology of Some Nagasaki Atomic-Bomb Casualties, Am. J. Ophth. **30**:127-135, 1947.

11. Cogan, D. G., and Donaldson, D. D.: Experimental Radiation Cataracts: I. Cataracts in the Rabbit Following Single X-Ray Exposure, A. M. A. Arch. Ophth. **45**:508-522 (June) 1951.

cortex at the equator. This is accompanied by (1) reorientation, and possible increase in amount, of cytoplasm with the formation of processes that straddle the equator; (2) loss of cell membrane, and (3) progressive changes in the nuclei, consisting of an initial increase in size, subsequent transformation from spherical to elliptical, and eventual increase in density and decrease in size, with ultimate disappearance of the nuclei. These changes are represented chronologically by their respective distances from the capsule. They are, as it were, a specialized and physiologically purposeful type of degenerative change whereby living cells are converted into a histologically inert hyaline substance.

Radiation induces morphologic changes in the entire anterior epithelium and characteristic changes at the equator. Instead of dropping off into the cortex, as is the normal custom of the equatorial cells, they pile up beneath the capsule, and some migrate beneath the capsule toward the posterior pole, where they tend to aggregate at the point of union of the lens sutures. The cells which are piled up at the equator, those which have migrated posteriorward and, to a less extent, those which have been damaged in situ on the anterior capsule may show a characteristic series of changes, which have been generally regarded as degenerative. It should be noted, however, that the regular swelling up of these cells ("balloon cells") and eventual bursting are not the usual manifestation of degeneration, and a somewhat different interpretation will be offered subsequently.

The early clinical manifestation of radiation cataract is a central opacity, which is generally stated to be in the posterior cortex, but the opacity is frequently described as having an unusually well-defined anterior face, and at least one group of authors, including one of us (D. G. C.),[12] has suggested that the initial opacity was in the capsule. While 3 of the 20 human lenses studied histologically did show capsular changes in the expected region, evidence in the rabbit leads to the unequivocal conclusion that, as Grzedzielski suggested for human beings, the early opacity is due to aberrant cells which have migrated beneath the posterior capsule. Flat preparations of posterior capsules show cells corresponding in degree and disposition to the opacities seen by the usual clinical means.[11] While adherent to the capsule, these cells may be removed by scraping; and the underlying capsule appears then of normal transparency. No flat preparations of human radiation cataracts have become available, but cells may be seen beneath the posterior capsule in cross section; and in at least two instances of radiation cataract in which an extracapsular cataract extraction was done, with liberal irrigation of the anterior chamber, a central plaque was left affixed to the posterior capsule in a region corresponding to the sharply demarcated opacity seen prior to the cataract extraction. In analogy to similar findings in radiation-induced lenticular changes in rabbits, it is assumed that this opacity was made up of aggregates of aberrant epithelial cells.

The factors involved in the migration of cells and their subsequent metamorphoses are far from clear; indeed, it is not apparent whether this migration is an active or a passive process. It is well known, however, that damage to cells

12. Cogan, D. G.; Martin, S. F., and Kimura, S. J.: Atom Bomb Cataracts, Science **110**:654-655, 1949.

exposed to radiation may be sufficient to prevent their full and normal differentiation but still be compatible with life of the cell and partial differentiation. If this principle is applicable to the lens cells, the piling up at the equator and subsequent curious changes in the cells, which have been interpreted simply as degenerative, may be looked upon as abortive attempts at formation of lens fibers. It is certainly true that the regular increase in cytoplasm of the cells, disappearance of the nucleus, and ultimate loss of the cell membrane that occur in the aberrant cells are analogous to what takes place at the equator normally. Moreover, these changes appear to be progressively a function of distance from the capsule, indicating a regular sequence of events, as is the case in the normal differentiation, rather than a spotty degenerative change. The chief difference between the changes in the aberrant cells beneath the posterior capsule and those of cells at the equator are the shape of the cells and their lack of transparency. These aberrant cells have abortive processes at best and show only a limited tendency to insinuate beneath adjacent cells, as do normal lens fibers. Their opaqueness may be due simply to their abnormal hydration, for with ordinary stains they show striking variability in density.

If these changes in the aberrant cells are interpreted correctly as abortive attempts at differentiation of lens fibers, the series of events may be reasonably clear. There is, first, a migration of isolated cells beneath the posterior capsule. These tend to aggregate in a region of least mechanical resistance, the junction of the posterior sutures. In swelling up, either the nucleus disappears before the cell bursts, leaving for a time only an eosinophilic sphere, or the cell bursts before the nucleus disappears, leaving free, floating nuclei in a milieu of unconfined cytoplasmic debris, producing a picture similar to that seen with the bursting of epithelial cells in bullous keratopathy.[13]

The changes in the anterior epithelium are somewhat similar. With severe irradiation there are fragmentation of the nuclei and death of the cells in situ. With less severe irradiation the cells appear to survive until the time of mitosis and then to produce aberrant mitoses and disintegration of the cells. But most interesting is the isolated occurrence of cells with an inordinate amount of cytoplasm, similar to the cells of the posterior capsule, and the occasional dropping-off of cells from the anterior capsule into the cortex, analogous to what was interpreted in the case of the posterior capsule as abortive attempts to form lens fibers.

Finally, it should be pointed out that many of these changes, and probably most, are not specific for radiation-induced cataracts. What appears to be specific for the radiation type is the early migration of cells beneath the posterior capsule, and possibly the occasional thickening of the posterior capsule, before any extensive changes have occurred in the cortex.

SUMMARY AND CONCLUSIONS

The clinical features of cataract in human beings characteristic of radiation are (1) the doughnut-shaped configuration, as seen with the ophthalmoscope, and (2) the sharply demarcated anterior boundary of the opacity and the bivalve configura-

13. Cogan, D. G.: Bullous Keratitis, with Particular Reference to Pathology of Experimental Corneal Vesiculation, Arch. Ophth. **25**:941-968 (June) 1941.

tion of the opacity, as seen with the slit-lamp biomicroscope. These are not seen in all stages of radiation cataract and are not exclusively found in radiation cataract, but when present they are strongly suggestive of the radiation type and are usually sufficient to differentiate the radiation type from posterior polar cataract and cataracta complicata, with which it may easily be confused.

The histologic changes of radiation cataract consist primarily of (1) failure of the cells at the equator to differentiate into lens fibers, and (2) early migration of cells beneath the posterior capsule toward the posterior pole. These aberrant cells may cause thickening of the capsule and undergo changes that are interpreted as abortive attempts at differentiation of lens fibers.

In general, the histologic manifestations of radiation cataract are less type-specific than are the clinical signs.

NONSURGICAL REPAIR OF STRICTURES OF THE LACRIMAL DRAINAGE SYSTEM

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TEMPLE, TEXAS

CONSTANT epiphora is a major annoyance to, if not a serious affliction of, many people. The results of treatment of epiphora, when due to partial or complete stenosis of the lower lacrimal canaliculus, are often less than desired. This is in contrast to the usually satisfactory results obtained in treatment of epiphora when due to dacryocystitis, closure of the nasolacrimal duct in infants, or ectropion. Epiphora due to partial stenosis of the lower canaliculus is much more frequent than that due to complete stenosis of this canal. Partial closure of the canaliculus is especially frequent in the older age group; often the puncta only are closed in these persons.

PARTIALLY STENOSED LOWER CANALICULUS

In 1949 I reported the use of a plastic rod for the treatment of partially stenosed lower lacrimal canaliculi.[1] Nylon rods [2] are now used instead of the plastic rods employed previously. There is no danger of breaking, and little, if any, tissue reaction, with nylon.

The lower canaliculus is first dilated until a No. 4 Bowman probe will pass. The nylon rod is molded into the approximate shape needed, and the point is rounded at one end. The rod is rigid enough to pass into the tear sac through the lower canaliculus. It is held in place on the cheek with adhesive (Fig. 1). Three to five days is sufficient time for it to remain in place.

Polyethylene tubing [3] is also satisfactory for use in cases of partial stenosis of the lower canaliculus. The tubing is inserted through a special thin-walled, 16- or 18-gauge needle.[4] These needles are made with blunted ends so that a false channel will not be created. It is necessary to dilate the lower canaliculus prior to insertion of the needle. The needle should be inserted with the stylet in place. The tubing

From the Department of Ophthalmology, Scott and White Memorial Hospital.

1. Veirs, E. R.: Surgical Treatment of Lacrimal Drainage System, Texas J. Med. **46**:111-113 (Feb.) 1950.

2. Nylon rods, one with outside diameter of 0.043 in. and the other with an outside diameter of 0.045 in., manufactured by the Clay-Adams Company, Inc., 141 E. 25th St., New York 10.

3. Manufactured by the Clay-Adams Company, Inc., 141 E. 25th St., New York 10.

4. B-D Special Needle, No. T462LNR, Becton, Dickinson & Co., Rutherford, N. J. The needles were described as "New Instruments" at the Fifty-Sixth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology, Oct. 14, 1951, Chicago.

is left in place three to five days, in the same way as the nylon rod. Polyethylene tubing with an outside diameter of 1.27 mm. will pass through the 16-gauge, special thin-walled needle, while tubing with an outside diameter of 0.965 mm. will pass through an 18-gauge, special thin-walled needle.

The results of treatment of partially stenosed lower canaliculi by the above methods are usually gratifying, in contrast to the disappointing results of repeated probings. It is obvious that if ectropion or a blocked nasolacrimal duct is also present, epiphora will not be stopped, even though the lower canaliculus is made patent.

The first patient treated by this method had had constant epiphora, owing to partial stenosis of the lower canaliculus as a result of use of radium in the nose for a malignant growth. For complete relief the plastic rod had to be reinserted the

Fig. 1.—Nylon rod or plastic tubing held in place with either adhesive tape or Scotch tape.

second time, several months after the original insertion, and left for three days. The procedure, however, has been used about three years, in many cases, and in only an occasional one has reinsertion been necessary.

COMPLETELY STENOSED LOWER CANALICULUS

Callahan [5] and Henderson [6] have each reported the use of polyethylene tubing for repair of the completely stenosed lower canaliculus. The method employed by each of them requires a surgical approach. Where complete stricture is encountered, the following method can be used in inserting a plastic tube to reestablish lacrimal drainage without an external incision. A small lacrimal probe is passed into the tear sac through the upper canaliculus, if it is patent. A sharp-

5. Callahan, A.: Surgery of the Eye: Injuries, Springfield, Ill., Charles C Thomas, Publisher, 1950, pp. 79-80.

6. Henderson, J. W.: Management of Strictures of the Lacrimal Canaliculi with Polyethylene Tubes, Arch. Ophth. **44**:198-203 (Aug.) 1950.

pointed, special thin-walled, 16- or 18-gauge needle, previously mentioned (one without the point being blunted), is used with its stylet in place. It is inserted through the lower punctum, or at the approximate site of the old punctum, and boldly pressed through the lower canaliculus (Fig. 2, left). The probe, if present in the upper canaliculus, is removed. The needle is then raised to a vertical position so that it can be passed through the nasolacrimal duct into the nose, in the same manner that a nasolacrimal duct is probed. The polyethylene tube is then passed through the lumen of the needle, and the needle is withdrawn, leaving the tubing in place (Fig. 2, right). Thiopental (pentothal*) anesthesia simplifies the procedure, although an injection of procaine combined with topical application of tetracaine in the nose, is satisfactory.

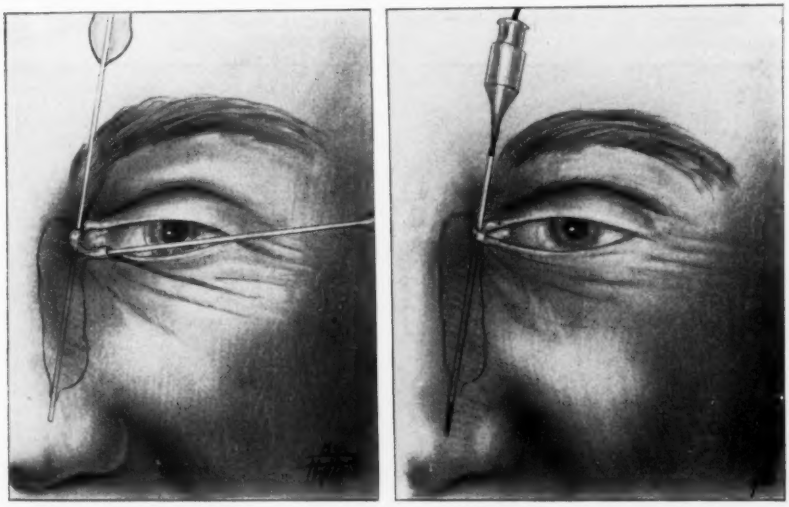

Fig. 2.—Left: special thin-walled needle with stylet making new lower canaliculus in a case of complete stenosis.

Right: needle probe withdrawn. The needle has been passed through the nasolacrimal duct into the nose, the plastic tube threaded through the needle, and the needle withdrawn, leaving the plastic tube in place.

A variation of the procedure is to pass the needle directly into the nose, bringing it out at the anterior tip of the middle meatus, instead of passing it through the nasolacrimal duct (Fig. 3). If this variation is used, the needle, after it has been passed through the scar tissue into the tear sac, is directed down and medially at an angle of about 75 degrees to the horizontal. Then, as before, the polyethylene tube is passed through the lumen of the needle and the needle withdrawn, leaving the tube in place. The tubing can be left in place almost indefinitely, with little irritation or inconvenience to the patient. It is left in place for at least 90 days.

After the plastic tubing is in place in the completely stenosed lower canaliculus, one can cut it off at the punctum. The soft tissues are "milked" toward the nose, exposing about 1 or 2 mm. of the tubing, which can then be cut off again. When

the tissues resume their normal position, the tubing will not show, and it will not scratch the eyeball. The patient will have almost no epiphora with the tubing in place, and it can be worn with few, if any, symptoms. The length of the tube in the nose should not be too great; otherwise, it may work out.

I have one patient who had complete stenosis of the inner half of the lower canaliculus, with a history of epiphora from childhood; she has worn the tubing for many months, with almost complete relief from the epiphora. Fluorescein can be seen in the nose after its instillation into the conjunctival sac. As long as she is free of symptoms, I shall leave the tubing in. This principle is similar to Callahan's except for the location of the tubing and a much simpler method of insertion.

STENOSED NASOLACRIMAL DUCT

In infants probing of the nasolacrimal duct is very successful in treatment of the obstructed nasolacrimal duct. Even with frank dacryocystitis probing will

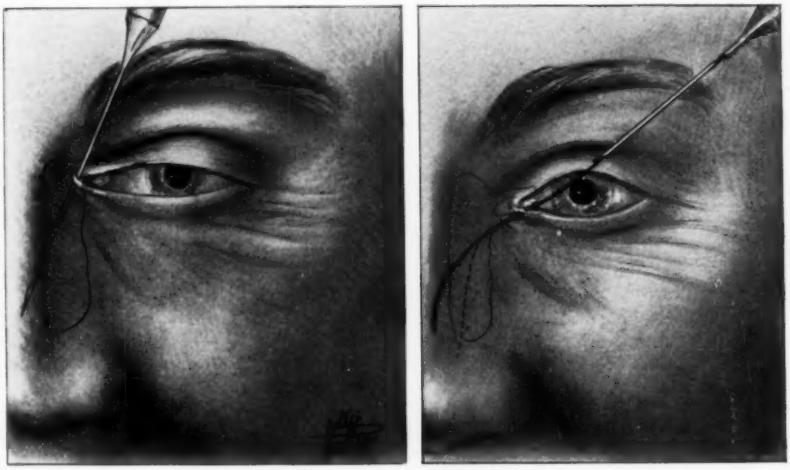

Fig. 3.—Special thin-walled needle passing directly into the nose, by passing the nasolacrimal duct. Plastic tubing is then threaded through the needle and left in place when the needle is withdrawn. This route apparently is as satisfactory as the one shown in Figure 2.

occasionally cure the stenosis. In patients other than infants probing of the stenosed nasolacrimal duct is rarely successful. Usually a dacryocystorhinotomy is performed on persons of the older group.

It was decided to try the use of plastic tubing in cases of the latter group. The lower canaliculus is first dilated and the blunted, special thin-walled needle, with the stylet in place, is passed into the tear sac and down through the nasolacrimal duct. The stylet is removed, and the plastic tubing is inserted through the needle, traversing the lower canaliculus, tear sac, and nasolacrimal duct. The needle is withdrawn, and the plastic tubing is held in place on the cheek with Scotch tape. In cases of stenosis of the nasolacrimal duct only, one week seems sufficient time for the tubing to remain in place. One injection of long-acting penicillin is given.

Rarely, one sees a patient with stenosis of both the nasolacrimal duct and the lower canaliculus. Repeated external incisions are often performed on such a patient. When stenosis of the lower canaliculus is at the junction of the tear sac and the canaliculus, as was seen in a case recently, the blunted needle can usually be forced into the tear sac. The needle is then passed through the nasolacrimal duct into the nose. It should be left in place from 60 to 90 days in such cases.

COMMENT

Many patients with partial stenosis of the lower lacrimal canaliculus are seen by the oculist. The treatment of these patients with the use of nylon rods or plastic tubing, as described here, is simple and satisfactory.

Relatively few patients are seen with complete stenosis of the lower canaliculus. In the majority of these patients the condition is the result of injury, that is, an injury that has cut through the canaliculus. Treatment of such a condition has been most unsatisfactory. It is hoped that the use of polyethylene tubing inserted through the thin-walled needle, as described, will make its treatment simpler and the results better. It seems preferable, in cases of complete stenosis of the lower canaliculus, to sever the polyethylene tubing just inside the punctum and leave it in place indefinitely. It is possible that even a year or more may be required for the new tract to become epithelized. In the meantime, the patient is comfortable and has little, if any, epiphora.

The nylon rod described above can also be inserted into an injured canaliculus immediately after an injury, that is, at the time the wound is sutured. This is far preferable to trying to make a new channel through scar tissue later.

The polyethylene tubing, when it is passed through the lower canaliculus, tear sac, and nasolacrimal duct, using the thin-walled, blunted needle, will effect cure in a large percentage of cases of dacryocystitis. It will require a large series of patients and many years to determine the percentage of cures which will be obtained. The procedure is simple, however, requiring only a little more time to perform than simple probing of the nasolacrimal duct.

Clinical Notes

NOTE ON POSTOPERATIVE MANAGEMENT OF RETINAL DETACHMENT

J. W. JERVEY, M.D.
GREENVILLE, S. C.

"TIME hath often cured the wound when reason failed to heal" (Seneca). However, it may be that in the postoperative care of retinal detachment we have relied too much on time and not enough on reason.

This clinical note calls attention to three cases of retinal detachment in which the period of postoperative bed rest was considerably shortened, with no untoward effects. My premise is that if healing is to follow surgical treatment of retinal detachment it will occur quickly, as in any clean injury. Furthermore, it will, and does, take place in spite of motion. The eye is not still in sleep at all times, or in closure, and cannot be splinted or stabilized by any acceptable method. There is also doubt, after the retina is back in place, as to whether any particular position is preferable.

The management in the first case here recorded was prompted by the fact that the patient was an old man with cardiac failure who could not lie down for any length of time. The aphakic glaucomatous eye was seen within two days of the onset of symptoms of detachment in the upper nasal quadrant. The tear was well visualized. The sclera was cauterized, fluid withdrawn, and the retina observed in place. Subretinal fluid and air were injected into the vitreous through the cornea and pupil, restoring firm tension to the globe. The patient was returned to his room in a wheel chair with eyes bandaged and was allowed to remain up when and as he chose, bathroom privileges being allowed at all times. The fundus was observed on the fifth day and the retina found in place. He was given peephole glasses on the sixth day and allowed to go home on the 10th day. A cure resulted, with retention of his former field and virtually normal central vision at the time of this report, now more than two years.

In the second case, that of a woman aged 53, a detachment was found in the upper temporal quadrant and operated on probably four to six weeks from the onset of symptoms. The tear was easily found. Simple cauterization and withdrawal of fluid were first done. The patient was put to bed, but no attempt was made to immobilize her. The eye was observed on the third day, and there was a small detachment lateral to the macula. This progressed rapidly and the eye was reoperated on on the eighth day, this time with the injection of subretinal fluid and air into the vitreous. The eye remained soft, and a pressure bandage was applied. On the third day after the second operation, the patient, who was restless, was allowed up in a chair and peephole glasses used. The retina could not be well seen on account of vitreous haze. On the 12th day, the retina being in place, the patient was allowed to return home. Three weeks later there had developed a subretinal hemorrhage in the macular area. Six months later the retina was in place and the field and tension were normal, but the central vision was only 20/200, not quite as good as prior to operation.

In the third case, that of a woman aged 69, a detachment in an aphakic eye in the upper temporal quadrant was operated on one week after the earliest symptoms of detachment were first observed. The tear was not found. Cauterization was done and 1 cc. of air injected through the pupil. On the fourth day the retina was found in place and the patient allowed up. On the eighth day she went home with the retina in place and the eyes bandaged. Two weeks later a small detachment far forward in the temporal area was observed. This progressed rapidly, and three days later a second operation was performed, with transpupillary replacement of subretinal fluid and injection of air to increase the pressure. On the third postoperative day the retina was in place and the patient allowed up in a chair. As tension was low, air was

again injected through the pupil to make the eye firm to palpation. She was allowed to go home on the fifth postoperative day with eyes bandaged. At the time of writing, three months later, the retina is still in place, the field full, and the vision normal.

COMMENT

These cases are not presented in detail, nor is it intended to argue that the procedures used are the best. However, they indicate two things: first, that the postoperative care of retinal detachment can, and should, be simplified to the great advantage of the patient mentally, physically, and financially, and, second, that the eye tolerates this type of surgical procedure exceedingly well, as shown by the very slight reaction even when a second operation was performed after a short interval.[1]

1. Personal communications have been received from Dr. Algernon Reese, of New York, and Dr. Francis Heed Adler, of Philadelphia, both of whom have had similar experiences with elderly patients on their services.

CARCINOMA OF BULBAR CONJUNCTIVA FOLLOWING CATARACT EXTRACTION

Report of a Case

DALTON OLIVER, M.D.
AND
WINSTON ROBERTS, M.D.
WINSTON-SALEM, N. C.

While carcinoma involving the bulbar conjunctiva at or near the limbus is not infrequent, it is of unusual interest when it occurs after cataract extraction as a complication thereof. Tumors of the bulbar conjunctiva are located most commonly temporally and within the palpebral aperture.[1] According to Benedict [2] and Ash and Wilder,[3] occurrence of these tumors is commonest in elderly men.

Because of the merging of two types of epithelium at the limbus, this area is the site of predilection for development of epitheliomas. As compared with carcinoma elsewhere, the usual epithelioma at the limbus is of relatively low malignancy and penetration. The transition between and clinical differentiation of inflammatory hyperplasia and true neoplasms is often vague.[4]

It has been suggested that injury frequently enters into the etiology of epithelioma of the limbus, and in many cases a history of previous trauma may be elicited.[5] Epithelioma has also been noted arising from a pterygium and developing from a simple papilloma. Spaeth [6] stated that simple papilloma not infrequently develops along the line of incision after muscle surgery.

The following case is presented as an instance of epithelioma developing directly as a postoperative complication of cataract extraction.

REPORT OF CASE

W. S. P., a white man aged 72, a retired furniture worker, was admitted to the North Carolina Baptist Hospital on June 14, 1950, with a diagnosis of bilateral mature senile cataract. On June 15 an intracapsular cataract extraction was performed on the left eye, and on June 19 a similar procedure was carried out on the right eye, without complication. The McLean type of corneoscleral sutures and a reflected conjunctival flap were used, and the conjunctival incisions were closed in each instance with two untied, continuous sutures of 6-0 black silk. These sutures were removed without difficulty on the seventh postoperative day. The postoperative course was smooth, and the patient was discharged June 29.

He was seen in the outpatient clinic at regular intervals and continued to improve. On Aug. 4, approximately five weeks after his discharge, a small, whitish yellow, warty mass was first noted in the right eye approximately 3 mm. from the lateral limbal margin at the 9-o'clock position.

From the Department of Ophthalmology, Bowman Gray School of Medicine.

1. Swan, K. C.; Emmens, T. H., and Christensen, L.: Experiences with Tumors of the Limbus, Tr. Am. Acad. Ophth. **52**:458-469 (May-June) 1948. Samuels, B.: Tumors of Conjunctiva and Lids: Brief Review, Arch. Ophth. **26**:789-796 (Nov.) 1941.

2. Benedict, W. L.: Epithelioma of the Limbus, S. Clin. North America **9**:813-822, 1929.

3. Ash, J. E., and Wilder, H. C.: Epithelial Tumors of the Limbus, Tr. Am. Acad. Ophth. (1941) **46**:215-222 (May-June) 1942.

4. Duke-Elder, W. S.: Text-Book of Ophthalmology, Vol. 2, St. Louis, C. V. Mosby Company, 1939, pp. 1775-1787.

5. Ash and Wilder.[3] Duke-Elder.[4]

6. Spaeth, E. B.: Principles and Practice of Ophthalmic Surgery, Ed. IV, Philadelphia, Lea & Febiger, 1948, p. 603.

Fig. 1.—Appearance of carcinoma of the bulbar conjunctiva.

Fig. 2.—Postoperative appearance of the eye.

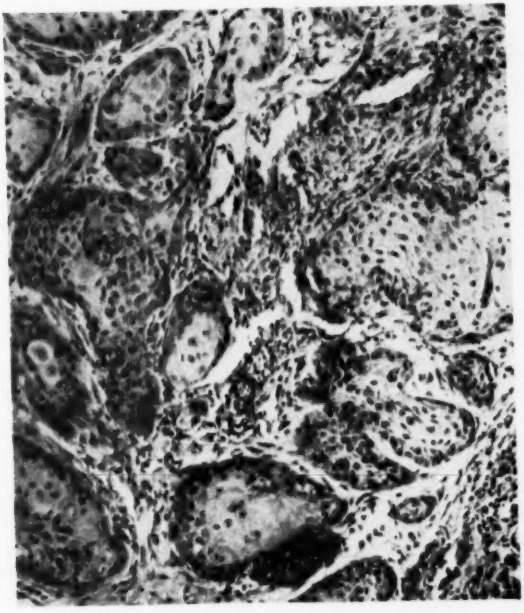

Fig. 3.—Epidermoid carcinoma of the bulbar conjunctiva; × 95.

This site corresponded to the inferior extremity of the arched conjunctival incision. Growth of the mass, which was at first thought to be a foreign-body granuloma, was rapid.

On Aug. 9 he was again admitted to the hospital. Figure 1 illustrates the appearance of the neoplasm at that time. The mass was warty in appearance, 4 mm. in diameter, firm, nontender, and adherent to the underlying tissues. With the use of local anesthesia, the mass was readily separated from the sclera and excised, and no evidence of extension noted.

Pathologic study of the specimen submitted revealed epidermoid carcinoma. The grade of the malignancy was designated as 1+. A photomicrograph (Fig. 3) illustrates the appearance.

The patient was followed subsequently at regular intervals. No radiation was administered. A recent photograph (Fig. 2) shows the area well healed with no evidence of recurrence. Only 10 months have elapsed since excision of the neoplasm, and the patient will be followed further for a possible later recurrence.

SUMMARY

Tumors of the bulbar conjunctiva are briefly discussed.

A case of epidermoid carcinoma of the conjunctiva developing in the healing conjunctival incision following a cataract extraction is presented.

Registry of Interesting Cases

It is the purpose of this section to record cases on the basis of interest only. No complete review of the literature is attempted and no claim for priority is implied except when specifically stated. Publication of case reports is subject to the concurrence of the Editorial Board on the interesting aspects of the case. It is requested that the reports submitted be as brief as is consistent with inclusion of the pertinent facts, bear a title that is as specific as possible, be prefaced by a paragraph that states why the author feels the case is of interest and be accompanied with photographs whenever possible.

PENETRATING ORBITAL WOUND WITH INTRACRANIAL COMPLICATIONS

We wish to report an interesting case of penetrating wound of the orbit with subsequent communication between the carotid artery and the cavernous sinus. It will be noted that extensive intracranial damage resulted, even though the original wound was slight and immediate ophthalmological and neurological changes were lacking. The ensuing neurological pattern was confusing and was clarified only at autopsy.

H. W., a Negro aged 27, was admitted to the University Hospital in an intoxicated state at 5 a. m. on Nov. 11, 1950, having been stabbed a few hours previously with a sharp instrument at the outer border of the left eyebrow. Examination in the emergency room revealed a superficial, irregular laceration ¾ in. (1.9 cm.) long, which seemed to be a minor injury, and it was very nearly decided to send the patient home. However, after some deliberation, he was finally admitted to the ophthalmology service for observation.

The extraocular movements were unimpaired, and the rest of the neurological examination revealed no abnormality. The blood pressure was 130/80; the pulse rate, 70, and the respiration rate, 24 per minute. During the 24 hours after his admission there gradually developed proptosis of the left eye with chemosis of the conjunctiva, periorbital and subconjunctival ecchymosis, and a dilated, fixed pupil (Fig. 1). There was no evidence of injury to the globe, media, or retina.

During the day after entrance to the hospital the patient remained somewhat lethargic, but this could have been attributed to his alcoholic intoxication. At the end of this time a neurosurgical consultant examined the patient, who could be aroused sufficiently to give fairly accurate responses. Rather pronounced nuchal rigidity was noted. Complete paralysis of the left third cranial nerve was found, and an audible bruit could be heard over the left orbit. Hypesthesia was elicited from the ophthalmic division of the left trigeminal nerve, and there was definite weakness of the right masseter muscle, with deviation of the jaw to the right. A left supranuclear facial palsy was observed. There was no diminution in the auditory acuity. The tongue deviated to the right side. Hypalgesia and thermalgesia were found on the left side of the body, but tactile sensibility and proprioception were intact. Motor power and coordination were good in all extremities, with no abnormalities in tonus. Reflexes were hypoactive in the upper extremities, but normally active and equal in the lower extremities, with an equivocal pyramidal-tract sign on the left. Routine roentgenograms of the skull revealed no abnormality. Lumbar puncture showed a frankly bloody spinal fluid, under normal pressure. The consultant's impression was that the patient had a carotid artery-cavernous sinus fistula with subarachnoid hemorrhage and that the hemorrhage might account for the great confusion in the neurological picture.

On November 12, a left internal carotid arteriogram was made, which demonstrated the suspected arteriovenous fistula between the carotid artery and the cavernous sinus (Fig. 2).

From the Section of Neurosurgery, Department of Surgery (Dr. Schneider), and the Department of Ophthalmology (Dr. Henderson), University Hospital.

Fig. 1.—Elevation of the ptosed left eyelid to show the third-nerve paralysis and the pronounced subconjunctival ecchymosis.

Fig. 2.—Left internal carotid arteriogram. The branches of the anterior and middle cerebral arteries are relatively well filled, in spite of the fact that some of the contrast medium has been diverted through a fistula into the signet-ring-shaped cavernous sinus (marked by the arrow).

With local anesthesia, the left internal carotid artery was then clamped off for one hour, and since there was no sign of vascular embarrassment to the left hemisphere, such as aphasia or right hemiparesis, the left internal carotid artery was ligated, and the bruit disappeared. For the next 12 hours the patient's condition was slightly improved. The left eyelid could be raised a bit higher, but he gradually became more lethargic, and 24 hours after ligation partial motor aphasia developed. This picture was maintained until November 17, when his condition progressed to total motor aphasia and he began to exhibit paradoxical weakness in the left arm. Another lumbar puncture at this time revealed a frankly bloody spinal fluid under a pressure of 350 mm. of water. It was thought that the patient was again bleeding intracranially and that further control of the lesion might be accomplished by applying a silver clip just proximal to the bifurcation of the left internal carotid artery. From our experience with intracranial aneurysms in the past, we have found that the ophthalmic artery has a much richer collateral supply than has been realized. It was anticipated prior to operation that this artery would also require occlusion in order that collateral flow into the cavernous sinus might be cut off.

On November 17 a transcoronal osteoplastic craniotomy was performed. The point of intracranial entry of the weapon above the left orbit could not be identified. Contrary to expectation,

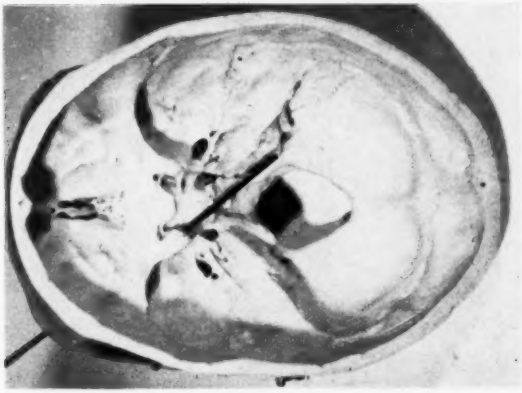

Fig. 3.—Probe showing the intracranial course which the weapon must have taken. The distance of penetration must have been at least 12 cm.

there was only a minimal amount of subarachnoid bleeding. The optic nerves and the chiasm were uninjured. The veins of the frontal cortex were dark blue and distended. Pulsation was absent in the internal carotid, anterior and middle cerebral, and anterior communicating arteries on the left side. The diameter of the right anterior cerebral artery was twice that of the left. Since thrombosis of the left internal carotid artery had already occurred after ligation of the vessel in the neck, nothing further could be accomplished. The brain in the left temporofrontal region was soft and necrotic. Closure of the wound was carried out, and the operation was well tolerated.

The medical consultant believed that the only therapy which might further aid the patient would be the prevention of propagation of the thrombus in the left internal carotid artery and its branches. Heparin therapy was begun and stellate blocks performed as desperate measures, with the realization that there might be increased bleeding at the site of the arteriovenous fistula. At 1:30 a.m. on November 19 the patient's blood pressure dropped and he died.

At necropsy slightly blood-tinged fluid was found in the intracranial subarachnoid space. There was a chip fracture of the lateral border of the left supraorbital ridge. The left frontal lobe was greatly softened anteriorly and laterally. Apparently, the instrument used for the

assault had been an ice pick, and it is possible to reconstruct the direction of its penetration by combining the neurological signs with the postmortem findings. The weapon entered just above the orbit at the lateral border of the left eyebrow and extended through the superior orbital fissure (Fig. 3), penetrating in succession the ophthalmic division of the left trigeminal nerve, the left carotid artery, and the cavernous sinus. The paralysis of the left third nerve was one of gradual development, so that it was probably due to the pressure from the expanding vascular fistula. As the weapon crossed the midline, it entered the pons to the left of the midline, cutting the motor nucleus of the trigeminal nerve. Beneath the surface of the pons, some of the fibers for the upper extremity lying in the medial portion of the right pyramidal tract were interrupted, causing paresis of the left arm. The lesion also included some of the fibers to the seventh nerve, causing a left supranuclear facial paresis. Farther back, the right lateral spinal spinothalamic

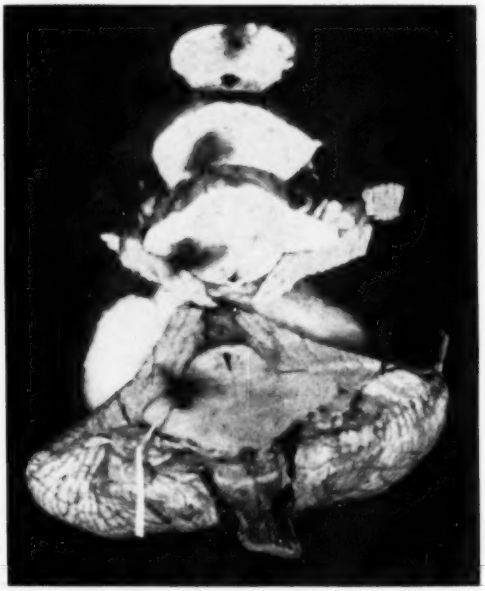

Fig. 4.—Serial sections demonstrating that the wound extended across the midline and had even penetrated the cerebellum.

tract was damaged, with subsequent hemihypalgesia and thermalgesia of the left side. Some of the fibers of the right 12th nerve were injured, causing the tongue to deviate to the right. In spite of the penetration of the lesion into the right cerebellar hemisphere (Fig. 4), no cerebellar signs could be demonstrated, but during the last few days of life the patient was unresponsive and could not be adequately tested. The distance from the wound of entrance, above the left orbit, to the farthest point of penetration must have been at least 12 cm. The cause of death was probably the extension of the pontine hemorrhage posteriorly toward the cardiac and respiratory centers, combined with extensive hypostatic pneumonia.

COMMENT

The severity of the wound was not sufficiently appreciated when the patient was seen in the emergency room, and he was almost sent home. The value of a complete

neurological examination is thoroughly demonstrated. The correlation of confusing neurological signs and final pathological findings is seen to be precise. Our failure to evaluate these signs properly was due at least partially to the fact that the extent of the wound was not fully realized and partially to a willingness to explain such bizarre findings on the basis of diffuse subarachnoid bleeding. It was found at necropsy that the patient had died of a combination of pontine hemorrhage and hypostatic pneumonia.

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JOHN WOODWORTH HENDERSON, M.D.

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PAPILLEDEMA ASSOCIATED WITH INCREASED INTRACRANIAL PRESSURE IN ADDISON'S DISEASE

Papilledema associated with increased intracranial pressure in patient with Addison's disease. Spinal fluid pressure determinations and ventriculography not undertaken. Ultimate disappearance of choking. Thrombosis of intracranial dural sinuses probable explanation for ocular signs.

L. S. (J.H.H.560011), a white woman aged 20, had been anemic for five years after severe pyelonephritis. She had complained of episodes of headache, associated with vomiting, and excessive weakness for five months before she was seen by us at the Johns Hopkins Hospital.

Three months prior to our examinations it had been recognized that the patient had Addison's disease (chronic adrenal insufficiency), and she had been given desoxycorticosterone acetate (percorten®). During this time, because of severe headaches with vomiting, she had been twice admitted to the hospital. On the last such admission she had been given two blood transfusions and had not evidenced any immediate reaction to the transfusion. Her eyegrounds were said to be normal at this time. Her condition became critical within a few days after the second transfusion. She was admitted to Johns Hopkins Hospital, in the service of Dr. Martin L. Singewald.

The patient was in good contact but critically ill. The bronzing of the skin and the mucous membranes of the mouth were classic. There were extreme weakness, headache, vomiting, and low blood pressure (90/60). The patient had no complaints with regard to the eyes, which appeared normal on external examination. Vision was 20/20—2 in each eye. Papilledema measured 3 D. in the right eye and 4 D. in the left eye. There were superficial, fresh-appearing exudates at the posterior pole of each eye and absence of hemorrhage. The veins were engorged. The arteries seemed normal. The fields were full, and there was pronounced enlargement of the blind spots.

In addition to desoxycorticosterone acetate, cortisone was administered.

Because of the evidence of increased intracranial pressure with "choking" of the optic disks, a ventriculographic study was desirable and lumbar puncture was contraindicated. However, Dr. J. E. Howard pointed to the extreme danger of ventriculography or any other surgical procedure in a patient with Addison's disease. Consequently, we decided to temporize on the basis that thrombosis of the intracranial dural sinuses was the probable etiologic factor.

At the end of four weeks of treatment with cortisone and desoxycorticosterone there was pronounced improvement. The headaches had disappeared. There was decided increase in strength. The blood pressure readings had become increased to near-normal. The papilledema had definitely lessened, so that it measured 2 D. in each eye; the fundus picture was otherwise unchanged. The patient was discharged to her home; unfortunately, she had a severe setback in a month, presumably due to a virus infection. Three months after she returned home there was slight residual blurring of the upper and lower margins of each disk. The exudates had disappeared.

Comment.—The occurrence of bilateral choked disk in a patient with Addison's disease is unique, at least in my experience. The possibilities that came to mind when the patient was first seen were (1) cerebral neoplasm, (2) venous-sinus thrombosis, dependent upon low blood pressure and hemoconcentration as a result of the transfusions, (3) tuberculoma, and (4) an allergic reaction to the transfusions. Dr. George W. Thorne added other possible explanations, namely, excessive desoxycorticosterone therapy and brain tumor with secondary adrenal insufficiency.

In retrospect, it would seem reasonable to assume that there had been thrombosis of the intracranial dural sinuses. With the development of compensatory circulation, the intracranial pressure returned to normal levels, and the papilledema disappeared.

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Annual Reviews

LIDS, LACRIMAL APPARATUS, AND CONJUNCTIVA

Review of the Recent Literature

JAMES H. ALLEN, M.D.
NEW ORLEANS

LIDS

Congenital Anomalies.—In a study of three families affected by congenital blepharoptosis, Musini [1] found that monomeric dominance existed. A remarkable hypoplasia of the levator palpebrae was found in all patients treated surgically. The ptosis was severer in later generations and when associated with other anomalies.

Friede [2] believes that the etiology of epicanthus can be determined only by phylogenetic studies. The medial epicanthus is specific for human beings. It is a phylogenetic relic without function and usually disappears in childhood. When it persists, anomalies of the nose, skull, and brain are present. For correction of this defect, Friede [3] advocates the implantation of fresh rib cartilage under the skin of the root of the nose.

Straith and Lewis [4] describe a family with dysostosis mandibularis. The mother and all her four children showed the defect in different degree. This occurrence is unusual, since the defect usually is sporadic.

Agnello [5] reports a case of bilateral congenital symmetrical epitarsus of the upper lid. Mattsson [6] observed ankyloblepharon filiforme adnatum in an otherwise healthy baby. Keizer and Ennema [7] describe dystropia of the medial canthus and of the puncta with blepharophimosis, rigid ptosis, broad flat nose, and flat ears in the first child of healthy parents. Bader [8] found the Meibomian glands missing in both lower lids of a 9-year-old girl. The hair follicles were clearly visible just under the conjunctiva.

1. Musini, A.: Contribution to the Study of the Hereditary Transmission of Congenital Blepharoptosis, Ann. ottal. e clin. ocul. **75**:45-54, 1949.

2. Friede, R.: Clinical Picture and Etiology of Epicanthus, Klin. Monatsbl. Augenh. **115**:12-27, 1949.

3. Friede, R.: Surgery of Epicanthus, Klin. Monatsbl. Augenh. **117**:178-186, 1950.

4. Straith, C. L., and Lewis, J. R.: Associated Congenital Defects of the Ears, Eyelids, and Malar Bones (Treacher Collins Syndrome), Plast. & Reconstruct. Surg. **4**:204-213, 1949.

5. Agnello, F.: A Case of Bilateral and Symmetrical Epitarsus, Gior. ital. oftal. **3**:385-390, 1950.

6. Mattsson, R.: Ankyloblepharon filiforme adnatum, Acta ophth. **28**:223-227, 1950.

7. Keizer, D. P. R., and Ennema, M. C.: Dystopia of the Medial Canthus and of the Puncta Lacrimalia with Blepharophimosis (van der Hoeve-Waardenburg), Nederl. tijdschr. geneesk. **95**:295-298, 1951.

8. Bader, A.: Congenital Aplasia of the Meibomian Glands in the Lower Lid, von Graefes Arch. Ophth. **150**:411-413, 1950.

Lid Movement.—Motion pictures of blinking [9] show that the lower lid remains almost stationary. The upper lid begins to close as a shade would drop, but the movement is completed by narrowing of the palpebral fissure in a zipper-like action from the lateral toward the medial canthus. This may aid in the displacement of foreign material toward the lacus lacrimalis. The left eye usually lags behind in closure and opens earlier, but this may be related to right- and left-handedness.

Zutt [10] describes three cases in which a residual inability to close the eyes on command persisted after the first serious symptoms of intracranial hemorrhage had cleared. In two cases this was associated with forced grasping with the hand. The author discusses the importance of a lesion in the right hemisphere in causing these symptoms.

Souter [11] observed a man aged 24 who for 12 years had been able to evert his upper lids, at first assisted by finger pressure on the temples; but later he was able to evert the right upper lid voluntarily, without finger aid.

Chalazion and Astigmatism.—Casanovas [12] found a patient with a chalazion of the left upper lid who had visual acuity of 0.4. Ophthalmometric examination revealed corneal astigmatism, which required —1.25 D., axis 175, to bring the visual acuity to normal. Three days after extirpation of the chalazion visual acuity began to improve, and in 14 days the astigmatism had disappeared, with the return of normal visual acuity without correction.

Ptosis.—Cameron [13] describes a familial ptosis occurring after the age of 40 in seven men and eight women, representing five generations. There were no associated extraocular or intraocular muscular anomalies, but the ptosis was progressive and did not respond to drugs usually effective in relief of myasthenia.

Cazzullo and Oxilia [14] found disturbances of the corticosubcortical electrical activity, but no cerebral focal lesion, in electroencephalograms of three patients who had congenital ptosis with cyclic oculomotor paralysis.

Jonkers [15] adds a new generation, with seven affected persons (four females and three males), to the pedigree reported by Flieringa. Two of the patients had unilateral ptosis, and, in addition, one had unilateral external ophthalmoplegia.

Wight [16] observed complete recovery from total ocular paralysis and ptosis due to the sting of an insect in an 8-year-old girl.

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9. Anantanarayana Ayer, A.: Note on the Mechanism of Eyelid Closure in Blinking, Proc. All-India Ophth. Soc. **10**:154-158, 1949.

10. Zutt, J.: The Impossibility of Keeping the Eyes Closed: Apraxia or Compulsive Movement, Nervenarzt **21**:339-345, 1950.

11. Souter, W. C.: Auto-Eversion of Upper Eyelid(s), Voluntary, Unaided, (Mimical Ectropion of Upper Lids, of S. Holth), Tr. Ophth. Soc. U. Kingdom **68**:207-212, 1949.

12. Casanovas, J.: Chalasion and Astigmatism, Arch. Soc. oftal. hispano-am. **9**:23-27, 1949.

13. Cameron, P. B.: Familial Ptosis of the Eyelids Appearing in Middle Life, Arch. Ophth. **43**:818-822 (May) 1950.

14. Cazzullo, C. L., and Oxilia, E.: Clinical and Physiological Observations on 3 Cases of "Ptosis a Bilancia," Ann. ottal. e clin. ocul. **76**:421-444, 1950.

15. Jonkers, G. H.: Familial Congenital Ptosis Combined with Other Motor Defects of the Eye, Nederl. tijdschr. geneesk. **2**:1471-1472, 1950.

16. Wight, J. F.: Ocular Paralysis with Ptosis Due to a Sting or Bite, South African M. J. **24**:65-66, 1950.

Ptosis Surgery.—Although Guy [17] is emphatic in the condemnation of all operations for ptosis which utilize the superior rectus, Marin Amat [18] believes that from both the anatomical and the physiological point of view it is better to use the superior rectus than the frontalis muscle. Therefore, except in the case of paralysis of the superior rectus, he uses Elschnig's modification of the Motais operation.

Berke [19] uses a modification of the Motais operation which he believes offers the following advantages: 1. The procedure may be used even though the superior rectus is paralyzed. 2. Slipping of the transplanted muscle tongue is reduced to a minimum. 3. The notch in the upper lid frequently produced by the original Motais procedure is replaced by a smooth curve. 4. Entropion is prevented. 5. A good lid fold is obtained. 6. Postoperative hypotropia is reduced. 7. The operation can be used in young children. 8. The lid moves vertically synchronously with the globe. He exposes the tarsus through a skin incision placed where the upper lid fold is desired and then incises the conjunctiva just above the tarsus. The superior rectus is exposed from the conjunctival approach after the insertion of a lid speculum. The tendon is divided into three longitudinal tongues, on each of which is placed a double-armed suture 2 mm. from the insertion of the tendon. The tongues are severed between the sutures, and the sclera and the superior rectus are shortened the desired amount by passing two double-armed 4-0 chromic surgical gut sutures through the lower lip of the conjunctiva, the tendon stump, the belly of the muscle at the desired position, and, finally, the upper lip of the conjunctiva. When these sutures are tied, the conjunctival wound is closed around three exposed tongues of muscle, 5 to 10 mm. long. The three tongues are pulled through the incision in the palpebral conjunctiva by their sutures and spread out on the upper surface of the tarsus. The six needles are passed between the tarsus and the orbicularis through the lid margin so that they come out in the gray line and with each pair 5 to 6 mm. apart. The skin incision is closed with silk sutures, which are passed through the transplanted tongues of the superior rectus tendon in order to anchor the skin to them and insure a good lid fold.

Steckler [20] reports enthusiastically on the Dickey operation, listing twelve advantages for it, the most important of which seems to be that the lid remains in the position in which it is fixed at the time of operation. On the other hand, Starke [21] believes that best results are to be expected from the method of Blaskovics, and Farina and De Toledo Piza [22] advocate a modification of the Wiener-Lexer technique.

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17. Guy, L. P.: Use of the Superior Rectus in Operations for Blepharoptosis, New York J. Med. **50**:2417-2420, 1950.

18. Marin Amat, M.: Superiority of Motais' Operation in Treatment of Ptosis of the Eyelid: A Few Clinical Cases, Arch. Soc. oftal. hispano-am. **9**:726-735, 1949.

19. Berke, R. N.: Motais-Parinaud Type of Operation for Ptosis: Report on 35 Cases, Arch. Ophth. **41**:324-333 (March) 1949; Operation for Ptosis Utilizing the Superior Rectus Muscle, Tr. Am. Acad. Ophth. **53**:499-518, 1949; Arch. Ophth. **42**:685-708 (Dec.) 1949.

20. Steckler, M. I.: Operations for Blepharoptosis, Arch. Ophth. **42**:283-291 (Sept.) 1949.

21. Starke, H.: Treatment of Ptosis, Med. Klin. **45**:399-400, 1950.

22. Farina, R., and De Toledo Piza, P.: Blepharoptosis: Blepharoplasty by a Modified Wiener-Lexer Technique, Plast. & Reconstruct. Surg. **5**:97-101, 1950.

Dermatitis.—Weekers,[23] Mejer,[24] and Brahy [25] describe many cases of contact dermatitis of the lids, sometimes associated with similar lesions of the fingers, in nurses or attendants who for two to six months had been preparing streptomycin injections for patients. Withdrawal from that duty or wearing of protective gloves resulted in cure.

Eczema of the lids may also be produced by various anesthetics, sulfonamides, atropine, penicillin, mercury oxycyanide, yellow or orange mercuric oxides, and many other substances, according to Sidi and Dobkevitch,[26] and Druault.[27]

Herzog [28] reports that chronic eczema of the lids with severe itching, even though little is visible externally, is often caused by lack of protein and fat in the diet. He obtained good results in these cases with vitamin therapy.

Gangrene.—Pages and Viennot-Bourgin [29] describe four cases of unilateral gangrene of both lids seen in a seven-year period. They believe the disease typical of North Africa or the tropics. They were unable to find a bacterial cause and refer to the hypothesis of noninfectious vascular lesions advanced by Morax and suggest as an alternative cause the thrombosing action of the poison of some insect peculiar to the region.

Blepharochalasis.—Lohr [30] describes the development of blepharochalasis in a 7-year-old boy who had had recurring edema of the lids and conjunctival and subcutaneous hemorrhages for three years.

Tumors of the Lid.—Polychronacos [31] reports the occurrence of clear cysts, probably arising from the glands of Moll, in the external canthus of both eyes of a patient aged 30.

Franceschetti and Stadlin [32] describe two cases of hidradenoma, benign pinhead-sized tumors of the skin of the lid arising from the sweat glands. In addition to the tumor of the lower lid, one patient had cornea guttata and subepithelial ring-shaped opacities. Both patients were young women.

In three cases of xeroderma pigmentosum, Magni [33] found several types of tumors of the lids.

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23. Weekers, L.: An Occupational Disease of Nurses: Eczema of the Eyelids Due to Streptomycin, Rev. méd. Liège **5**:320-322, 1950.

24. Mejer, F.: Allergic Eczema of the Lid as an Occupational Disease Caused by Streptomycin, Wien. klin. Wchnschr. **61**:702-703, 1949.

25. Brahy, J.: Blepharo-Conjunctivitis and Streptomycin, Union méd. Canada **78**:472, 1949.

26. Sidi, E., and Dobkevitch, S.: Palpebral Lesions Caused by Treatment, Arch. opht. **9**:311-320, 1949.

27. Druault, T. N.: Eczema of the Eyelids, Concours med. **71**:1341-1342, 1949.

28. Herzog, H.: Chronic Eczema of the Eyelids as a Symptom of Chronic Undernourishment, Klin. Monatsbl. Augenh. **117**:653-654, 1950.

29. Pages, R., and Viennot-Bourgin: Gangrene or Putrid Necrosis of the Eyelids, Maroc méd. **27**:478-482, 1948.

30. Lohr, K.: A Case of Blepharochalazia in Childhood, Klin. Monatsbl. Augenh. **115**:28-31, 1949.

31. Polychronacos, D.: Symmetrical Cysts of the Eyelids, Bull. Soc. hellen. ophth. **17**:229-230, 1950.

32. Franceschetti, A., and Stadlin, W.: Hidradenoma of the Lids, Ophthalmologica **120**:3, 1950.

33. Magni, S.: Ocular Manifestations in Xeroderma Pigmentosum (Clinical and Histologic Details), Boll. ocul. **29**:84-96, 1950.

Biopsy of a tumor of the left upper lid, which spread rapidly to the right upper lid, in a woman aged 67 showed the lesion to be a lymphocytic lymphosarcoma with leukemia, according to Levitt.[34]

Fuchs [35] reports eight cases of divided nevi of the lids. Divided nevi are situated on the upper and lower lids in such a manner that they appear to be one lesion when the lids are closed. The author warns against operation on nevi if no malignant lesion is present.

Esente [36] removed a cylindromatous tumor of the lacrimal gland by superciliary orbitotomy in a man aged 21.

Among 1,000 patients, Schwab [37] found 40 with syringoma, or epithelioma adenoides cysticum. The tumors were small, rounded, firm, yellowish-tinged, and almost invariably multiple. Often similar tumors were found on the neck or thorax. They were commoner in women but were never seen before puberty.

Carcinoma of the Meibomian glands in the experience of Muthayya [38] is the commonest tumor affecting the eyelid, the cases forming 3.68% of his series. It has a predilection for the upper lid and affects women oftener than men. The average age of involvement is 48. The tumor begins as a firm, ill-defined nodule, which soon ulcerates and later extends. Rice and Lindeke [39] emphasize the importance of histologic examination of all growths which clinically resemble a recurrent chalazion.

De Castro [40] found 56 cases of cancer of the lid among 1,439 cases of cancer of the face. The youngest patient was 7 years of age, but 12% of the patients were under 30, 51% from 31 to 60, and 36% over 60, the oldest being 87.

Of 45 carcinomas of the lids, Lisch [41] found that 77.8% were of basal-cell and 22.2% of squamous-cell type. He considers surgical resection the method of choice.

During the 12-year period from 1933 to 1944, Stetson and Schulz [42] treated 301 patients with carcinoma of the eyelids. In 76% of all patients radiation therapy was used, but in only 11 did the tumor prove resistant. Several methods of x-ray therapy were used, and some patients were treated with radon. Seventy-one patients were treated surgically. The incidence of failure of the initial treatment was 13%; however, in all but 5 of the 29 cases of failure the growth was controlled by subsequent management. Apparent cures were obtained in 97.5%. No significant difference was noted in the percentage of failures following the various methods of treatment. The cause of failure was inadequate irradiation or inadequate surgical intervention. Failure to respond to primary treatment was twice as frequent with

34. Levitt, J. M.: Lymphosarcoma of the Lid and Conjunctiva, New York J. Med. **50**:2315-2316, 1950.

35. Fuchs, A.: Divided Nevi of the Eyelids, Urol. & Cutan. Rev. **54**:88-90, 1950.

36. Esente, I.: Contribution to the Study of Cylindromatous Tumours of the Lacrimal Gland, Gior. ital. oftal. **2**:193-213, 1949.

37. Schwab, F. I.: Syringoma or Epithelioma Adenoides Cysticum of the Eyelids, von Graefes Arch. Ophth. **150**:388-404, 1950.

38. Muthayya, R. E. S.: Meibomian Carcinoma, Proc. All-India Ophth. Soc. **10**:70-71, 1949.

39. Rice, M. L., and Lindeke, H. I.: Adenocarcinoma of the Meibomian Gland, Am. J. Ophth. **33**:1434-1437, 1950.

40. de Castro, M.: Cancer of the Eyelid, Resenha clín.-cient. **17**:344-346, 1948.

41. Lisch, K.: Carcinoma of the Eyelids, Krebsarzt **4**:217-218, 1949.

42. Stetson, C. G., and Schulz, M. D.: Carcinoma of the Eyelid: Analysis of 301 Cases and Review of the Literature, New England J. Med. **241**:725-732, 1949.

squamous-cell as with basal-cell lesions. Complications of treatment were not numerous, being mostly undesirable side-effects of irradiation, such as stenosis of the lacrimal duct, in which the lesion involved the inner canthus; this occurred in 31 cases. The authors do not think that routine use of radium is justified.

Del Regato [43] reports three-year cures in 78% of 168 cases of carcinoma of the skin of the lids following roentgen therapy.

Villanova, Oller Coromians, and Rubio [44] describe a basal-cell epithelioma of the inner canthus and conjunctiva in a woman aged 75. The tumor was treated with radium, but a nodule persisted, even after repeated irradiation. Upon removal, it proved to be a spindle-cell epithelioma. The authors think that the difference between the two histologic pictures was due to maturation and metaplasia brought about by the application of radium.

Lid Surgery.—In cases with a temporary problem of exposure of the cornea, as in coma, postoperative lagophthalmos, or transitory facial paralysis, Cogan [45] has found duco® cement useful. The patient's lids are closed, and the lashes of the upper lid are glued to the lower lid by application of the cement, time being allowed for each layer to dry between applications. The medial ends of the lids are left free, to permit flow of tears and drainage from the conjunctiva.

In three cases of severe blepharospasm, Barré Rohmer, and Philippides [46] found it necessary to sever branches of the orbicular portion of the seventh cranial nerve. They believe the procedure should be reserved for severe cases but found that the orbicularis muscle was still capable of voluntary contraction, in spite of some degeneration, in their three cases.

Herzum [47] reports that readhesion of the lid margins after a canthotomy may be prevented by suturing a small U-shaped roll of cellophane® between the lids at the outer angle.

Butler [48] has found that the excision of an inverted wedge from the tarsus has restored the normal position of the lids in cases of spastic entropion. He makes a 3 mm. incision through the conjunctiva and tarsal ligaments along the lower border of the tarsal plate, separates the tarsal plate from the orbicularis with blunt-tipped scissors up to the follicles of the cilia, and then excises a wedge of conjunctiva and tarsus, with the apex just missing the palpebral margin. The incision is closed at a right angle to the lid margin with silk or surgical gut.

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43. Del Regato, J. A.: Roentgen Therapy of Carcinoma of the Skin of the Eyelids, Radiology **52**:564-573, 1949.

44. Villanova, X.; Oller Coromians, F., and Rubio, J.: Basal- and Spindle-Cell Epithelioma of the Angle of the Eye, Actas dermo-sif. **40**:910-912, 1949.

45. Cogan, D. G.: A Simple Method for Temporary Closure of the Lids, Arch. Ophth. **42**:188 (Aug.) 1949.

46. Barré, J. A.; Rohmer, F., and Philippidès, D.: Successful Surgical Treatment of Severe Blepharospasm: Remarks on the Paradoxical Behavior of the Orbicularis Palpebrae, Rev. neurol. **82**:134-136, 1950.

47. Herzum, K.: A Procedure for Lengthening the Palpebral Opening, Klin. Monatsbl. Augenh. **115**:73-74, 1949.

48. Butler, J. B. V.: A Simple Operation for Entropion, Arch. Ophth. **40**:665-667 (Dec.) 1948.

Although Rossler [49] reports continued success in cases of spastic entropion by section of the lateral palpebral ligament, followed by application of a horizontal entropion suture, Hartleib [50] warns of danger unless the suture is secured in the temporal region with adhesive tape. Schopfer,[51] however, believes that a suture properly applied in a transverse direction through the eyelid is adequate in most cases.

Davids [52] has found that in his hands a slight modification of von Graefe's operation has been most successful in cases of senile entropion.

Nataf and Hebert,[53] in a review of procedures for the correction of trichiasis, emphasize the importance of straightening the angles of the lids. Henry [54] advocates the total excision of the tarsus in cases of trachomatous trichiasis. He found it necessary to make a canthoplasty also in approximately 20% of the cases. Trabut [55] expressed similar views and described a special plate which facilitates eversion of the lid in these cases.

Hatschek [56] advises the removal of a wedge-shaped piece of tarsal conjunctiva from the nasal position of the lower lid in case of slight ectropion of the lower lid but warns that this is ineffective for total or cicatricial ectropion.

In cicatricial ectropion, Gonzalez Ulloa [57] emphasizes the importance of extension of the raw surface after section of the adhesions before applying a dermoepidermic graft. Malbec and Beaux [58] advocate the use of whole-thickness grafts from the supraclavicular region for this purpose. However, Forsius [59] found that the coarser skin of the chest gave better results in a case of ichthyosis congenita with ectropion.

In a review of treatment of deformities of the eyelids, Figi [60] emphasized the importance of immediate repair of injuries of the lids if the patient's condition permits. The essential basic principles to be observed are careful cleansing, accurate

49. Rossler, F.: Entropion Suture Following Ligamentotomy, Klin. Monatsbl. Augenh. **115**:395-400, 1949.

50. Hartleib, R.: Temporary Suture in Acute Entropion, Klin. Monatsbl. Augenh. **116**:311, 1950.

51. Schopfer, O.: A Simple Operation for Curing Spastic Entropion, Klin. Monatsbl. Augenh. **115**:40-42, 1949.

52. Davids, H.: Contribution to the Surgery of Senile Entropion, von Graefes Arch. Ophth. **150**:420-421, 1950.

53. Nataf, R., and Hebert, C.: Surgical Treatment of Trichiasis, Rev. internat. trachome **26**:188-194, 1949.

54. Henry, L.: Resection of the Tarsus and Canthoplasty in the Operation for Trichiasis, Arch. opht. **9**:708-721, 1949.

55. Trabut, G.: Entropion-Trichiasis in North Africa: Its Operation by the Conjunctival Route, Arch. opht. **9**:701-707, 1949.

56. Hatschek: A Simple Method of Surgical Treatment in Ectropion of the Lower Lid, Klin. Monatsbl. Augenh. **115**:555-556, 1949.

57. Gonzalez Ulloa, M.: Correction of Cicatricial Ectropion by Means of a Skin Graft Transplanted on an Artificially Distended Area, Rev. brasil. cir. **18**:573-584, 1949; Medicina, México **29**:409-419, 1949.

58. Malbec, E. F., and Beaux, A. R.: Cicatricial Ectropion of the Eyelids, Prensa méd. argent. **36**:2458-2461, 1949.

59. Forsius, H.: Ectropion Operations in Ichthyosis Congenita, Acta ophth. **27**:573-579, 1949.

60. Figi, F. A.: Treatment of Deformities of the Eyelids, Proc. Staff Meet., Mayo Clin. **25**:330-333, 1950.

apposition of the margins of the wound, and the application of a firm pressure dressing.

For minor defects of the skin of the lids, Thiersch grafts or free full-thickness grafts of hair-free skin are used by Figi,[60] Heinsius,[61] Dzorova,[62] and Suarez Villafranca.[63] For larger defects, sliding grafts or pedicle flaps are used. Figi uses the Dupuy-Dutemps operation for replacement of a third to a half of a lid. Heinsius uses Kreibig's modification of Fricke's flap for a total substitution of the eyelids. Böck[64] implants ear cartilage in the portion of Fricke's flap which eventually will be used for forming the lid. However, Safar[65] and Böck agree that the Wendell-Hughes procedure is the best method for repairing a total defect of the lower lid.

In an excellently illustrated report on plastic reconstruction of lid and socket, Scarborough and Landers[66] emphasize the following points: use of general anesthesia, cutting of the grafts large enough to allow for both immediate retraction and late shrinkage, perfect hemostasis, avoidance of tension on sutures, and a minimum of five days before changing the dressing.

LACRIMAL APPARATUS

Congenital Anomalies.—Sjögren and Eriksen[67] relieved keratoconjunctivitis sicca in a 5-year-old girl with congenital complete absence of tearing by obliterating the puncta.

Although supernumerary lacrimal puncta are encountered rarely, Carreras Matas,[68] Straub,[69] and Bottasso[70] each report a case; and Sédan[71] reports five cases in one family (grandfather, two sons, and two grandsons). Two of Sédan's patients also had a fistula of the lacrimal sac.

Ferraris de Gaspare[72] describes two cases of congenital lacrimal fistula, and Faldi,[73] one case with two fistulae of one sac.

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61. Heinsius, E.: Experiences in Plastic Surgery of the Eyelids, Klin. Monatsbl. Augenh. **115**:35-40, 1949.

62. Dzorova, N. S.: Reconstruction of Eyelids and Conjunctival Sac, Ophth. Bull. Moskva **28**:36-37, 1949.

63. Suarez Villafranca, M. R.: Contribution to Blepharoplasty According to the Free-Graft Technique, Arch. Soc. oftal. hispano-am. **10**:851-860, 1950.

64. Böck, J.: Plastic Substitution of the Lower Eyelid, Wien. med. Wchnschr. **99**:375-376, 1949.

65. Safar, K.: Simultaneous Plastic Repair of the Two Eyelids in the Course of an Amplified Wendell L. Hughes Operation, Wien. klin. Wchnschr. **61**:713-714, 1949.

66. Scarborough, C. P., and Landers, P. H.: Plastic Socket and Lid Reconstruction, Am. J. Ophth. **33**:1269-1274, 1950.

67. Sjögren, H., and Eriksen, A.: Alacrimia Congenita, Brit. J. Ophth. **34**:691-694, 1950.

68. Carreras Matas, M.: Supernumerary Lacrimal Punctum, Arch. Soc. oftal. hispano-am. **10**:393-396, 1950.

69. Straub, W.: A Curious Deformity of the Inferior Canaliculus, Klin. Monatsbl. Augenh. **114**:563-565, 1949.

70. Bottasso, G.: Congenital Anomalies of the Puncta and Inferior Lacrimal Canaliculi, Rass. ital. ottal. **19**:94-100, 1950.

71. Sédan, J.: Supernumerary Lacrimal Ducts and Congenital Fistula of the Lacrimal Sac in Five Members of the Same Family, Ann. ocul. **182**:855-858, 1949.

72. Ferraris de Gaspare, P.: Complete and Incomplete Congenital Fistula of the Lacrimal Sac, Boll. ocul. **27**:739-745, 1948.

73. Faldi, S.: Twofold Lacrimal Monolateral Congenital Fistula, Gior. ital. oftal. **3**:222-227, 1950.

A patient with an entirely normal nasolacrimal system was found to have an accessory nonfunctional, though patent, second system, beginning in the form of a fistula 3 mm. below the inferior canaliculus and opening into the inferior meatus (Delaney and Oliver [74]).

A case of chronic tearing caused by congenitally ectopic puncta is presented by Keeney.[75]

In a series of 1,000 consecutive full-term infants, Kendig and Guerry [76] report 57 cases of congenital impatency of the nasolacrimal duct. They advise applications of penicillin ointment three times daily and massage of the sac but resort to probing if the obstruction is not relieved in six months. Von Herrenschwand [77] also advises simple pressure on the sac, with care to prevent the fluid in the sac from escaping into the conjunctiva while applying the pressure. However, Koke [78] favors irrigation of the lacrimal sac and early probing, with use of local or no anesthesia. He reports no ill effects in 116 infants treated by this method. The obstruction was relieved by irrigation in 14, by a single probing in 88, and by two or more probings in 11. Only 3 of the 116 infants were not cured.

Sykowski [79] believes that the prophylactic instillation of silver nitrate solution may cause dacryocystitis. He cites the cases of 3 infants and refers to 10 other infants who had dacryocystitis or nasolacrimal obstruction, all of whom had silver nitrate prophylaxis. However, the only suggestion of proof was based upon the reinstillation of a drop of silver nitrate solution into an eye in which nasolacrimal obstruction had not been relieved. Three days later irrigation of the lacrimal sac produced white, stringy, clumpy debris, an experience which is common in the irrigation of an obstructed nasolacrimal sac at that interval, even without the instillation of silver nitrate. Kendig and Guerry state that there is no reason to incriminate the Credé procedure as the cause of impatency. This is proved in the studies being made on substitutes for silver nitrate in prophylaxis, since the incidence of obstruction of the nasolacrimal passages in the newborn is not reduced by any of the drugs.

Tears.—Henderson and Prough [80] found that the flow of tears decreased roughly as the age increased when values for patients of successive decades were compared. The decrease in mean values, however, was not marked, and probably was without clinical significance. Among the younger patients, females exhibited a greater flow of tears than males; and in the group older than 60 the reverse was found, although the difference was slight. In the main, however, sex was not a factor in the flow of tears. Approximately 32% of patients were found to have a difference in the flow

74. Delaney, A. J., and Oliver, S. H.: Accessory Membranous Nasolacrimal Duct: Report of a Case, U. S. Armed Forces M. J. **1**:336-339, 1950.

75. Keeney, A. H.: Congenital Anomaly Associated with Absence of the Lacrimal Papillae and Causing Chronic Tearing, Am. J. Ophth. **32**:1586-1588, 1949.

76. Kendig, E. L., Jr., and Guerry, D., III: Incidence of Congenital Impatency of the Nasolacrimal Duct, J. Pediat. **36**:212-213, 1950.

77. von Herrenschwand, F.: Treatment of Congenital Lacrimal Obstruction, Österreich. Ztschr. Kinderh. **5**:189-194, 1950.

78. Koke, M. P.: Treatment of Occluded Nasolacrimal Ducts in Infants, Arch. Ophth. **43**:750-754 (April) 1950; Tr. Pacific Coast Oto-Ophth. Soc. **30**:57-65, 1949.

79. Sykowski, P.: Silver Nitrate Causing Dacryocystitis, Am. J. Ophth. **32**:1127-1128, 1949.

80. Henderson, J. W., and Prough, W. A.: Influence of Age and Sex on Flow of Tears, Arch. Ophth. **43**:224-231 (Feb.) 1950.

of tears from their eyes. However, there was no significant difference in the rate of flow observed between the hours of 8 a. m. and 5 p. m.

Using the method of Meyer and Hahnel, Regan [81] determined the lysozyme content of human tears formed reflexly in response to a lacrimator (benzyl chloride). Variations of ±33% of the mean were found in normal subjects. In such conditions as acute conjunctivitis, corneal ulcer, iridocyclitis, dacryoadenitis, and atrophy of the lacrimal gland the lysozyme content of the tears was found to be lower than in the normal state.

Vancea and Marculescu [82] report that after implantation of preserved skin, according to Filatov's method, the initial lacrimal elimination of sodium chloride is slightly increased, but immediately this increase is followed by an extremely pronounced and prolonged decrease. They suggest that this observation might be developed into an objective method for estimation of the biogenic action of implants of preserved tissue.

Two cases of crocodile tears accompanied by the Türk-Stilling syndrome were observed by d'Ermo,[83] and a probable case of Roger's blue disease (ventricular septal defect) in which red tears often occurred was reported by Angelone.[84]

All causes of lacrimation are considered by Traquair [85] in a short systematic survey, which includes a discussion of established methods of treatment.

McLaughlin [86] advises that operative measures for the correction of tearing in facial paralysis should provide support of the cheek and corner of the mouth by transplantation of muscle and tendon; correction of the lid deformity, preferably by lateral tarsorrhaphy; dilation of the punctum, and diathermic reduction of the caruncle.

Kaufman [87] thinks that epiphora in middle-aged patients due to constriction of the puncta is commoner than is indicated by the literature. He cites two cases of this type in which long-standing epiphora was relieved by dilation of the puncta and irrigation of the lacrimal sac.

Concretions.—Nine faceted concretions were removed from one patient by Riddell.[88] Microscopic examination of a crushed concretion showed Streptothrix, but there was no evidence of calcium carbonate in the x-ray diffraction pattern of another of the concretions.

Gutzeit [89] removed a concretion weighing 30 mg. from the canaliculus of a laborer who had worked with chalk and mortar for many years.

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81. Regan, E.: The Lysozyme Content of Tears, Am. J. Ophth. **33**:600-605, 1950.

82. Vancea, P., and Marculescu, C.: Effect of Implants of Preserved Tissue (Filatov's Method) on the Lacrimal Elimination of Sodium Chloride, Arch. opht. **10**:212-216, 1950.

83. d'Ermo, F.: Two Cases of Crocodile Tears of Congenital Nature Accompanied by Türk's Syndrome, Boll. ocul. **28**:273-288, 1949.

84. Angelone, L.: Red-Colored Tears Associated with Roger's Disease, Ann. ottal. e clin. ocul. **75**:287-294, 1949.

85. Traquair, H. M.: Classification of Epiphora, with Remarks on Diagnosis and Treatment, Tr. Ophth. Soc. U. Kingdom **67**:169-174, 1948.

86. McLaughlin, C. R.: Epiphora in Facial Paralysis, Brit. J. Plast. Surg. **3**:87-95, 1950.

87. Kaufman, S. I.: Atresia of the Lachrymal Puncta, Eye, Ear, Nose & Throat Month. **29**:494, 1950.

88. Riddell, W. J. B.: Streptothrix Colonies in the Lacrimal Canaliculus: Report of a Case, with a Note on Diffraction Spectrum Examination, Glasgow M. J. **30**:452-454, 1949.

89. Gutzeit, R.: Solitary Stone in Lower Lacrimal Duct in a Man with Bilateral Zonular Cataract, Klin. Monatsbl. Augenh. **116**:77-78, 1950.

Bilateral suppurative dacryocystitis developed in a patient aged 47 with lymphatic leukemia. Busina [90] thought that leukemic infiltrations, resulting in compression of the lacrimal ducts, caused the trouble.

Lacrimal Conjunctivitis.—Theodore [91] emphasizes the possibility that obstinate conjunctivitis is due to a subclinical infection of the canaliculus, and Capolongo [92] reports that biomicroscopic examination of the conjunctiva in diseases of the lacrimal apparatus sometimes reveals changes simulating trachoma. The reviewer has had similar experiences and makes it a rule to investigate the lacrimal apparatus routinely in cases of inflammatory disease of the conjunctiva.

Rhinosporidiosis.—Rambo,[93] from observations on eight cases of rhinosporidiosis of the lacrimal sac, reports that the lesion is slow in onset and characterized by unilateral noninflammatory, extensive, painless edema of the lower lid, giving the appearance of early elephantiasis. Treatment consists of excision of the sac with its mass of granulation tissue, followed by tight packing of the cavity for four or five days.

Tumors.—Ten years after excision of a lacrimal sac, a firm, elastic, walnut-sized tumor developed between the inner canthus and the root of the nose just above the medial canthal ligament. At operation Schlagenhauff [94] found a mucous retention cyst of the anterior ethmoidal cells.

A lacrimal sac removed by Toselli [95] showed plasma-cell infiltration of the thickened sac wall and necrosis of the epithelium.

A case of Mikulicz' syndrome with keratitis sicca was found by Kaufman [96] to be due to Hodgkin's disease.

Five cases of mixed tumor of the lacrimal gland are reported by Vrabec [97] (two), Froboese,[98] Oxilia,[99] and Fleming.[100] The tendency of such tumors to recur is exemplified by the cases of Froboese and Fleming.

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90. Busina, K.: Bilateral Phlegmonous Dacryocystitis in Course of Leukemia, Československ. ofth. **6**:221-223, 1950.

91. Theodore, F. H.: Streptothrix as a Cause of Follicular Conjunctivitis and Other Obscure Conjunctivitides, Am. J. Ophth. **33**:1225-1230, 1950.

92. Capolongo, G.: Biomicroscopic Examination of the Conjunctiva in Diseases of the Lacrimal Apparatus, Gior. ital. oftal. **3**:424-432, 1950.

93. Rambo, V. C.: Rhinosporidiosis of the Lacrimal Sac, Proc. All-India Ophth. Soc. **10**:72-76, 1949.

94. Schlagenhauff, K.: Mucocele of Ethmoidal Cells in Region of the Lacrimal Sac, Wien. klin. Wchnschr. **61**:716-718, 1949.

95. Toselli, C.: Plasmoma of the Lacrimal Sac, Rass. ital. ottal. **18**:333-337, 1949.

96. Kaufman, S. I.: An Early Manifestation of Hodgkin's Disease: Report of a Case, Eye, Ear, Nose & Throat Month. **29**:257-258, 1950.

97. Vrabec, F. I.: Myxochondroepithelioma in the Region of the Lacrimal Gland, Československ. ofth. **5**:203-217, 1949; Important Histological Details in "Mixed" Tumors of the Lacrimal Gland, Ophthalmologica **120**:210-218, 1950.

98. Froboese, C.: Fibromyxochondroepithelioma ("Endothelioma") Adenomatosum Cylindromatosum (Mixed Tumour) of the Lacrimal Gland, Which Continued to Recur for Several Years, von Graefes Arch. Ophth. **150**:490-508, 1950.

99. Oxilia, E.: Epithelial Tumors of the Lacrimal Gland, Ann. Ottal. e clin. ocul. **76**:227-237, 1950.

100. Fleming, N.: Adeno-Carcinoma (Mixed Tumour) of the Lacrimal Gland, Brit. J. Ophth. **33**:763-769, 1949.

Surgery.—A new method of closing the lacrimal canaliculi is presented by Sysi.[101] He makes an incision in the skin of the lid 2 mm. from, and parallel with, the lid margin and then introduces a probe into the canaliculus, detaching it from the surrounding tissue. He cuts the canaliculus just below the punctum and as far nasally as possible, removing the intervening segment. He completes the procedure by closing the incision with a few silk sutures and destroying the punctum by diathermy.

Morgenstern [102] cites 17 cases to point out the advantages of the use of electrocoagulation in treatment of eversion of the puncta; in recanalization of closed puncta and canaliculi, hypertrophic mucous membrane, and other growths which block the portion of the nose into which the nasolacrimal duct drains, and in destruction of scar tissue of the upper half of the nasolacrimal duct. He thinks it especially useful because it eliminates bleeding.

In a case of epiphora in a girl who had the lower punctum and half of the lower canaliculus obliterated in a skiing accident, Gayer Morgan [103] obtained relief by slitting the upper canaliculus in its whole length and inserting a wool wick for three days. Four other patients were treated in the same manner; two of them have been comfortable for over six years. In a sixth patient, requiring a dacryocystorhinostomy, a lower punctum was created by inserting a probe from the sac through the lower canaliculus to the point of obstruction and making the incision onto the end of the probe. The opening was maintained by passing the probe through the new opening, bending the end over onto the lid, and cutting off the opposite end in the lacrimal sac. The probe was left in place for one week; then the opening was dilated, and the passage into the nose was irrigated every few days. Three years after the operation the new opening was still functioning.

Henderson [104] found that polyethylene tubing, 1 mm. in outside diameter and 0.6 mm. in inside diameter, could be used to reestablish the patency of an obstructed canaliculus. The canaliculus is dilated, and the polyethylene tubing is inserted to the sac and sutured in place. The method is also suitable for the reconstruction of a cut or torn canaliculus. The tubing is left in place for varying lengths of time, depending on the circumstances.

Scharf [105] reports the use of a "supramid" tube for the recanalization of the nasolacrimal duct. The anterior wall of the lacrimal sac is opened; then the nasolacrimal duct is opened, and a "supramid" tube of proper diameter is inserted. The sac is closed with surgical gut. Irrigations are started on the fifth day. The "supramid" tube is well tolerated and heals into the tissues without being absorbed.

Hallum [106] outlines the history of operations on the lacrimal sac from the time of Celsus to the present day. Most recent authors agree with him that dacryocysto-

101. Sysi, R.: Suggestion for New Method of Closing the Lacrimal Canaliculi, Acta ophth. **27**:409-411, 1949.

102. Morgenstern, D. J.: Electrocoagulation for Cure of Chronic Tearing and Pertinent Added Related Considerations, Am. J. Ophth. **33**:893-903, 1950.

103. Gayer Morgan, O.: Stenosis of the Lower Canaliculus, Brit. J. Ophth. **34**:113-114, 1950.

104. Henderson, J. W.: Management of Strictures of the Lacrimal Canaliculi with Polyethylene Tubes, Arch. Ophth. **44**:198-203 (Aug.) 1950.

105. Scharf, J.: Procedure to Recanalize the Nasolacrimal Duct with Implantation of a Supramid Tube, von Graefes Arch. Ophth. **150**:678-682, 1950.

106. Hallum, A. V.: Dupuy-Dutemps Dacryocystorhinostomy, Am. J. Ophthal. **32**:1197-1206, 1949.

rhinostomy has supplanted dacryocystectomy, but there is considerable variation in method. Hallum bases his procedure on the Dupuy-Dutemps technique and reports relief in all but 1 of 55 patients with an adequate follow-up observation. Welt [107] reports successful results in 95.4% of cases. Meyer,[108] using Toti's operation (dacryocystorhinostomy), reports success in 45 of 52 cases, whereas Kanbaj [109] notes complete recovery in 95 of 97 patients with a similar intranasal procedure. Successful modifications or simplifications of the operation are described by Montanelli [110]; Caballero del Castillo [111]; Pichette, Audet, and Gelinas [112]; Goncalves Duarte and Ramos [113]; Ruedi,[114] and Alger.[115] Bonham [116] advocates the intranasal operation because an external incision is not required and the normal relations of the sac are not disturbed. Eriksen [117] reports a case of meningitis developing the day after a Toti operation, in which treatment with penicillin and "lucosil" (sulfamethylthiodiazole) was successful. The youngest patient to undergo this operation among those covered by this review was reported by Redmond [118]—a 3-year-old child.

Gunther [119] uses an endoscope 14 mm. in length and 4 mm. in diameter to examine the nasal opening after a Toti operation. Shortly after the operation the opening is wide and covered with blood and mucus, but with healing the opening becomes smaller and the blood disappears. Granulation tissue may develop and block drainage in two to four weeks after the operation; however, it can be removed easily with a wire loop. In old cases with blockage, complete closure of the nasal mucous membrane is usually observed. This does not contraindicate reoperation, which is generally successful. Beiras Garcia [120] reports similar studies, with use of a urethroscope.

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107. Welt, B.: Dacryocystorhinostomy: Report of 150 Cases of Dacryocystitis Occurring from 1938 to 1947, Arch. Otolaryng. **51**:83-95 (Jan.) 1950.

108. Meyer, F. W.: Surgery of the Lacrimal Apparatus, Klin. Monatsbl. Augenh. **116**:79-80, 1950.

109. Kanbaj, G. G.: Treatment of Dacryocystitis by Endonasal Route, Vestnik oto-rino-laring. **2**:35-57, 1951.

110. Montanelli, M.: Simplified Method of Dacryorhinostomy, Gior. ital. oftal. **3**:293-310, 1950.

111. Caballero del Castillo, C.: New Contributions to Dacryocystorhinostomy, Arch. Soc. oftal. hispano-am. **10**:249-255, 1950.

112. Pichette, H.; Audet, J., and Gelinas, C.: Dacryocystectomy and Intranasal Fistulization, Laval méd. **15**:31-42, 1950.

113. Goncalves Duarte, W., and Ramos, T. A.: Technical and Instrumental Contribution to Dacryocystitis Surgery, Hospital, Rio de Janeiro **35**:423-435, 1949.

114. Ruedi, L.: New Methods of Carrying Out Old Operations, Pract. oto-rhino-laryng. **11**:415-424, 1949.

115. Alger, L. J.: Hemostasis in Tear-Sac Operations, Am. J. Ophth. **32**:845-847, 1949.

116. Bonham, W. L.: Intranasal Dacryocystotomy, South. M. J. **42**:477-480, 1949.

117. Eriksen, A.: Meningitis After Toti's Operation, Acta ophth. **27**:287-289, 1949.

118. Redmond, K. B.: Dacryocystorhinostomy in an Infant, Med. J. Australia **1**:462, 1949.

119. Gunther, G.: Dacryostomy-Rhinoscopy, von Graefes Arch. Ophth. **150**:405-410, 1950; New Apparatus for Investigation of Rhinostomy Opening After Toti's Operation, Klin. Monatsbl. Augenh. **114**:259-260, 1949.

120. Beiras Garcia, A.: Provisional Information on "Dacryoscopy," Arch. Soc. oftal. hispano-am. **10**:35-46, 1950.

Vannas [121] has obtained good results in cases of intractable epiphora by removal of the papebral portion of the lacrimal gland. He makes a skin incision 10 to 12 mm. in length, straddling the outer orbital margin just above the upper margin of the external canthal ligament. With a lacrimal speculum in the wound, the lacrimal gland is exposed by blunt dissection with forceps and carefully dissected from the conjunctiva of the fornix, which is held away by a suture or forceps.

In cases of severe xerophthalmos, Vannas [122] has made a partial transplantation of tissue of the normal lacrimal gland from a patient with epiphora. Moderate benefit was derived in one of three eyes. Experimental grafting of the lacrimal gland in rats has been less successful, often leading to severe complications. The experimental data suggest that injury to the excretory duct in the course of the procedure is chiefly responsible for the poor results, but there is evidence of moderate regeneration of the duct. Thus, Vannas has hope that better results may be obtained with perfection of technique.

CONJUNCTIVA

Congenital Anomaly.—A fold of bulbar conjunctiva adherent to the limbus was observed as a congenital anomaly by Dorello.[123]

Conjunctival Flora.—Aznar [124] emphasizes the importance of making smears and cultures from the conjunctiva at the onset and at the height of inflammation.

Linhart [125] demonstrates that patching of an eye for 24 hours usually increases the bacterial flora. Average counts usually are increased threefold.

Vascular Phenomena.—Robertson, Wolf, and Wolff [126] found widespread sludging in the conjunctival vessels of normal, healthy subjects. It was produced by vasodilatation and could be reversed by the local use of vasoconstrictors. In the conjunctiva, sludging occurred as the result of an increase in the volume of the local vascular beds and a decrease in the velocity of blood flow.

McCulloch and Pashby [127] describe two types of aneurysms occurring in the conjunctival vessels—berry aneurysms and fusiform dilatations. The fusiform aneurysm is probably postinflammatory or arteriosclerotic. Berry aneurysms were found in 55% of all diabetic patients and in 14% of nondiabetic patients.

Acute Conjunctivitis.—Acute epidemic catarrhal conjunctivitis in Macedonia in 1946, 1947, and 1948 was caused by Pneumococcus and the Koch-Week bacillus.

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121. Vannas, M.: Removal of Palpebral Portion of Lacrimal Gland Through the Skin, Am. J. Ophth. **33**:354-356, 1950.

122. Vannas, M.: Transplantation of Lacrimal Glands in Man and Rat: Effect of Injury of Lacrimal Duct in Rat, Clinical and Experimental Study, Arch. Ophth. **43**:804-812 (May) 1950.

123. Dorello, U.: A Case of Conjunctival Anomaly, Gior. ital. oftal. **4**:74-80, 1951.

124. Aznar, A.: Clinical Importance of Conjunctival Flora, Cons. gen. col. méd. España, Bol. **9**:7-9, 1950.

125. Linhart, R. W.: Effect of Eye Patch on Organisms of the Conjunctival Sac, Am. J. Ophth. **33**:1280-1282, 1950.

126. Robertson, H. S.; Wolf, S., and Wolff, H. G.: Blood "Sludge" Phenomenon in Human Subjects: Notes on Its Significance and on Effects of Vasomotor Drugs, Am. J. M. Sc. **219**:534-537, 1950.

127. McCulloch, C., and Pashby, T. J.: Significance of Conjunctival Aneurysms in Diabetics, Brit. J. Ophth. **34**:495-505, 1950.

Milorad [128] reports that the greatest morbidity was in children. Instillation of penicillin drops (5,000 U. per cubic centimeter) every two hours reduced the duration of the infection from seven to two days.

Davis and Pittman [129] found that a severe form of acute conjunctivitis in the lower Rio Grande Valley of Texas was due to a bacillus of the genus Hemophilus. Streptomycin therapy was effective.

Several cases of severe acute conjunctivitis in St. Louis were found by Moor [130] to be due to Bacterium anitratum. Streptomycin therapy was effective.

Membranous Conjunctivitis.—A case of true membranous conjunctivitis is described by Oksala.[131] The lesion appeared shortly after birth in an otherwise healthy girl and healed spontaneously in approximately one year without complications. The cause was not established.

Saubermann [132] was able to save the affected eye in a case of conjunctivitis pseudomembranacea chronica granulomatosa. The disease usually affects children from 1 to 10 years of age. It is characterized by the formation of thick, diphtheritic-like pseudomembranes. A papillary hypertrophy, which may develop into granulomatous tumors, underlies the membranes. The cornea is infiltrated and may form an abscess and perforate. The cause is unknown.

Deep Conjunctivitis.—Dededimos [133] describes deep conjunctivitis as uniform dense hyperemia of the deep conjunctival vessels with or without slight secretion in the morning, the sensation of a foreign body, and slight periorbital pain. He lists the types as metastatic gonococcic conjunctivitis; rheumatoid conjunctivitis; scrofulous, electrical conjunctivitis; the conjunctivitis of firemen, resulting from intense heat; the conjunctivitis resulting from extension or suppuration from an adjacent sinus, and the type which appears during the recovery stage of contagious diseases.

Gonococcic Conjunctivitis.—Hugonot [134] describes a small epidemic of extragenital gonococcic conjunctivitis in a ksar of South Oran, Algeria. The disease was transmitted directly or indirectly by soiled linen.

Mineev [135] reports that six babies with gonococcic conjunctivitis (ophthalmia) were cured in five days without local treatment. The mothers, all of whom had genital gonorrhea, were given 15 gm. of sulfapyridine in five days and also were cured. The infants were breast-fed and apparently received enough sulfapyridine in the milk.

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128. Milorad, C.: Epidemic of Catarrhal Ophthalmia in Macedonia in 1946-1947 and in East Serbia in 1948, Voj.-san.-pregled **6**:429-430, 1949.

129. Davis, D. J., and Pittman, M.: Acute Conjunctivitis Caused by Hemophilus, Am. J. Dis. Child. **79**:211-222 (Feb.) 1950.

130. Moor, W. A.: Possible Occurrence of Bacterium Anitratum in Several Cases of Conjunctivitis, Am. J. Ophth. **32**:1593-1594, 1949.

131. Oksala, A.: A Case of Conjunctivitis Membranacea, Acta ophth. **28**:179-185, 1950.

132. Saubermann, G.: Conjunctivitis Pseudomembranacea Chronica Granulomatosa (Lignosa), Ophthalmologica **118**:666-686, 1949.

133. Dededimos, P.: Deep-Seated Conjunctivitis, Bull. Soc. hellen. opht. **17**:244-245, 1950.

134. Hugonot, R.: Localized Epidemic of Gonorrheal Conjunctivitis in a Saharan Environment, Arch. Inst. Pasteur Algérie **28**:95-102, 1950.

135. Mineev, P.: Treatment of Gonoblennorrhea of the Newborn by Absorption of Sulfathiazole from Mother's Milk, Vestnik oftal. **28**:42-43, 1949.

Prophylaxis of Gonococcic Conjunctivitis of the Newborn.—Several authors (Candano and associates [136]; Halbron and associates [137]; Berwind [138]; Sacks-Wilner and Sacks-Wilner,[139] and Dessy [140]) report series of newborn infants treated with varying strengths of penicillin ointment or solution as a substitute for the Credé prophylaxis. In Berwind's series, 1 case of gonococcic conjunctivitis developed in 1,090 newborn infants.

Dvorak,[141] on the basis of his own experience and published reports, concludes that penicillin could be used under controlled conditions but that silver compounds should be used outside institutions. However, Rejchert [142] opposes the use of penicillin even in maternity hospitals because of the difficulty of having to administer the ointment or drops several times a day for three to five days, because of the danger that penicillin-resistant strains of the organism will develop, and because of the risk of the development of an allergy to penicillin. Other authors mention the increased cost as an objection.

Bickel [143] compared the results of 30% sodium sulfacetimide drops, 10% sodium sulfacetimide ointment, and 1% silver nitrate drops in three groups, comprising approximately 600 infants. He considered 30% sodium sulfacetimide as effective as the silver nitrate. Watts and Gleich [144] report that the incidence of ophthalmia neonatorum decreased at Harlem Hospital when sulfathiazole was given orally as an adjunct to silver-nitrate prophylaxis.

Phlyctenular Conjunctivitis.—Missiroli and Oricchio [145] believe that phlyctenular disease may be regarded as an allergic phenomenon of tuberculosis because in their series of 90 children of ages up to 12 years with phlyctenular conjunctivitis, 55 (61%) had other tuberculous lesions. Of 375 children of the same age treated for pulmonary or glandular tuberculosis, 85, or 22% had phlyctenular conjunctivitis. The average sensitivity to tuberculin of the patients with phlyctenular

136. Candano, A.; Avila Cisneros, I., and Arelano, J. F.: Prophylaxis Against Ophthalmia Neonatorum: Penicillin as a Substitute for Credé's Method, Bol. Soc. med. centro materno-infantil **1**:57-61, 1950.

137. Halbron, P.; Lepage, F., and Mawas, H.: Prophylaxis of Ophthalmia Neonatorum by Penicillin, Arq. brasil. oftal. **12**:123-125, 1950.

138. Berwind, T.: Prophylaxis of Ophthalmia Neonatorum with Penicillin, Geburtsh. u. Frauenh. **10**:312-317, 1950.

139. Sacks-Wilner, A., and Sacks-Wilner, E. P.: Penicillin as a Prophylactic Against Ophthalmia Neonatorum, Arch. Ophth. **41**:444-449 (April) 1949.

140. Dessy, B.: Contribution to the Prophylaxis of Ophthalmia Neonatorum: Prophylaxis by Penicillin Substituted for Credé's Method, Giorn. batteriol. e immunol. **40**:441-454, 1949.

141. Dvorak, V.: Is the Prophylaxis by Credé's Procedure Still Necessary? Českoslov. oftal. **6**:173-176, 1950.

142. Rejchert, B.: Is Penicillin a Suitable Prophylactic in the Newborn? Českolov. oftal. **6**:176-177, 1950.

143. Bickel, J. E.: Sodium Sulfacetimide for the Prophylaxis of Gonorrheal Ophthalmia Neonatorum: Preliminary Report, J. Pediat. **37**:854-857, 1950.

144. Watts, S. G., and Gleich, M. M.: Penicillin-Silver Nitrate Prophylaxis Against Gonorrheal Ophthalmia of the Newborn: Preliminary Report on Use of Penicillin and Silver Nitrate Combined and of Silver Nitrate Alone, J. A. M. A. **143**:635-637 (June 17) 1950.

145. Missiroli, G., and Oricchio, D.: Etiology and Pathogenesis of Phlyctenular Conjunctivitis, Ann. Ist. Carlo Forlanini **12**:108-116, 1949.

conjunctivitis was higher than that of the group of tuberculous children without phlyctenulosis. However, Björkenheim [146] believes that, in addition to tuberculosis, streptococcic infection is also involved, for in a series of 122 patients with phlyctenulosis the antistreptolysin titer was found increased in 70.5%, whereas it was increased in only 13.3% of a control series consisting of 30 patients with external ocular diseases and 66 patients with refractive errors. In addition, beta-hemolytic streptococci were cultured from pharyngeal swabs in 37.5% of the patients with phlyctenulosis, but from none of the control series. Therefore, the author thinks that a streptococcic infection in a tuberculous patient may produce phlyctenulosis.

Capolongo [147] reports phlyctenulosis in 644 patients with measles, 360 with scarlet fever, 183 with chickenpox, 177 with pertussis, and 4,900 with typhus. He thinks that phlyctenulosis may often represent a hypersensitivity to the infectious agent.

Bonavolonta and Auricchio [148] report the successful use of streptomycin in the treatment of 20 patients with phlyctenular keratoconjunctivitis. The dose they recommend is 0.02 to 0.03 gm. per kilogram of body weight per day. Frolov [149] also reports success with streptomycin in 26 patients; however, he used drops of a solution of 1 gm. of streptomycin in 10 ml. of isotonic sodium chloride solution. The drops were administered every two hours for two or three days; then after an interval of two days the instillations were repeated for two days. However, he advises that the drops be used early, before deep ulcers form. Poleff [150] points out that penicillin is effective in phlyctenular or allergic disease only when the disease is caused by an organism against which the drug is bacteriostatic.

Conjunctival Tuberculosis.—Conjunctival tuberculosis (Pascheff [151]) may be divided into exogenous and endogenous forms. The exogenous form, usually ulcerative, is described as the conjunctival-glandular-febrile syndrome. The endogenous type appears in various forms—ulcerative, papillary, follicular or nodular, miliary, and tuberculomatous.

Kamel [152] believes that primary tuberculosis of the conjunctiva is rare, since only 33 cases were seen in the ophthalmic hospitals of Egypt from 1925 to 1948. The lesion was located in the palpebral conjunctiva in 48% of the cases, in the bulbar conjunctiva in 40%, and in the fornix in 12%. Males were involved more frequently (70%) than females. Only 4 of the 33 patients were more than 20 years of age, and 1 was 4 years old.

146. Björkenheim, B.: Pathogenesis of Eye Phlyctenules with Special Reference to the Role of β-Hemolytic Streptococci, Acta ophth. **27**:437-454, 1949.

147. Capolongo, G.: Interpretation of Phlyctenular Keratoconjunctivitis Which Appears in Some Infectious Diseases, Boll. ocul. **29**:441-456, 1950.

148. Bonavolonta, A., and Auricchio, B.: Streptomycin in the Treatment of Phlyctenular Keratoconjunctivitis, Pediatría **58**:539-549, 1950.

149. Frolov, G. F.: Treatment of Scrofulous Affections of the Eye with Streptomycin, Vestnik oftal. **29**:30-32, 1950.

150. Poleff, L.: Allergic and Microbial Pathogenesis of Phlyctenular Keratoconjunctivitis, Ophthalmologica **117**:338-342, 1949.

151. Pascheff, C.: Clinical and Histological Forms of Conjunctival Tuberculosis and Perifocal Keratitis, Compt. rend. Acad. belg. sc. **2**:57-59, 1949.

152. Kamel, S.: Primary Tuberculosis of the Conjunctiva, Brit. J. Ophth. **34**:322-327, 1950.

In reporting three cases of primary tuberculosis of the conjunctiva, Starke [153] emphasizes the importance of etiologic diagnosis in cases of the conjunctival-glandular syndrome. He found the pathologic organisms in glandular material more easily than in conjunctival material.

Several patients with conjunctival tuberculosis have been treated successfully with streptomycin. In their case of bilateral conjunctival ulcer, Das Gupta and Usman [154] used drops 12 times a day, combined with intramuscular injections of 1 gm. of the drug twice a day, for 4 days, followed by administration of 1 gm. daily for 21 days. Similar successes were reported by Sykowski,[155] Kamel,[152] and Donegan [156]; however, smaller intramuscular doses were used. Mata Lopez [157] found streptomycin given in association with other antibiotics and substances such as glucosulfone sodium (promin®), thiazolsulfone (promizole®), and vitamins, to be useful in treatment of all forms of ocular tuberculosis, including conjunctival lesions.

Epidemic Keratoconjunctivitis.—The usual incubation period of epidemic keratoconjunctivitis is 7 to 10 days (Thygeson [158]) but may vary from 5 to 19 days (Pellitteri and Fried [159]). The onset is acute; within 48 hours the eyelids, conjunctiva, caruncle, and plica semilunaris have become edematous; simultaneously, a painful swelling of the preauricular lymph node has occurred (Houllegatte [160]). The exudate is predominantly mononuclear and does not contain significant bacteria. Inclusion bodies are not found in scrapings (Pellitteri and Fried [159]). The conjunctival involvement may vary from follicular hypertrophy to severe pseudomembranous, and occasionally to membranous, conjunctivitis (Thygeson [158]). The acute phase of the conjunctival lesion usually lasts 8 to 12 days and subsides into a chronic inflammation, which persists from several weeks to as long as 18 months or more in some cases (Croll and Croll [161]). Small round subepithelial opacities develop in the cornea 7 to 10 days after the onset in most cases (Thygeson [158]) but may occur as late as 20 days after the onset (Houllegatte [160]). The corneal lesions reach their peak at about the fifth or sixth week (Croll and Croll [161]), then slowly

153. Starke, H.: Primary Tuberculosis of the Conjunctiva, Klin. Monatsbl. Augenh. **114**:107-112, 1949.

154. Das Gupta, B. K., and Usman, M.: Bilateral Symmetrical Tuberculous Ulcers of the Bulbar Conjunctivae Treated with Streptomycin, Brit. J. Ophth. **33**:501-505, 1949. Das Gupta, B. K.: Streptomycin in Treatment of Tuberculous Ulcers of the Conjunctiva: A Further Communication, ibid. **34**:681-683, 1950.

155. Sykowski, P.: Streptomycin Treatment of Conjunctival Tuberculosis, Am. J. Ophth. **33**:1291-1292, 1950.

156. Donegan, J. M.: Primary Tuberculosis of the Conjunctiva, Am. J. Ophth. **33**:1117-1121, 1950.

157. Mata Lopez, P.: Antibiotic Therapy of Tuberculosis of the Eye, Arch. Soc. oftal. hispano-am. **9**:415-423, 1949.

158. Thygeson, P.: Epidemiology of Epidemic Keratoconjunctivitis, Am. J. Ophth. **32**:951-959, 1949.

159. Pellitteri, O. J., and Fried, J. J.: Epidemic Keratoconjunctivitis: Report of a Small Office Outbreak, Am. J. Ophth. **33**:1596-1599, 1950.

160. Houllegatte: Epidemic Keratoconjunctivitis, Méd. d'usine **11**:139-143 1949; Epidemic Keratoconjunctivitis, Arch. mal. profess. **10**:260-261, 1949.

161. Croll, M., and Croll, L. J.: Epidemic Keratoconjunctivitis, Grace Hosp. Bull. **28**:55-60, 1950.

disappear. Visual loss may be severe at the peak of corneal involvement (Houllegatte [160]), but extremely good to normal vision usually returns. In only a few cases a slight diminution of vision does persist. Houllegatte emphasizes that the cornea never becomes vascularized. Offret and Albahary [162] report corneal involvement in only 8 of their series of 67 patients, an incidence which is in considerable contrast to the 95 to 98% reported by most previous authors.

In making a diagnosis of epidemic keratoconjunctivitis, such diseases as acute herpetic keratoconjunctivitis, acute follicular conjunctivitis, nummular keratitis, inclusion blennorrhea, and trachoma must be considered and excluded (Thygeson [158]).

Epidemic keratoconjunctivitis is a highly communicable disease, probably due to resistance of the virus to drying and dilution (Thygeson). Transmission within families is common, but the first-aid office and the ophthalmologist's office may be more important points of spread of the disease (Houllegatte [160]). In one instance it appears that 18 cases of office transmission occurred as the result of examination and treatment of one patient. In addition, four instances of transmission from a patient to a member of his family occurred in this small outbreak. However, simple control measures in the office broke the spread. The especially dangerous objects of transfer seem to be fingers, tonometers, solutions such as tetracaine or homatropine, contaminated masks, goggles, and common tools.

Early treatment with convalescent serum and aureomycin apparently modified the disease in cases reported by Correard and Plessier.[163] Houllegatte [160] believes that 50 ml. of convalescent serum given before the fifth day prevents corneal complications in 60% of cases. Sulfonamides and penicillin do not alter the course of the disease (Croll and Croll [161]; Offret and Albahary [162]). Capalbi [164] reports success with aureomycin in several cases, with chloramphenicol in several others, and with the intravenous, oral, and local use of paraaminobenzoic acid. Bietti [165] reports that penicillin, streptomycin, tyrothricin, and aureomycin were not effective.

Acute Epidemic Follicular Conjunctivitis.—Hubert and Lebas [166] describe a mild, transient epidemic of acute follicular conjunctivitis type Béal frequently associated with pharyngitis in workers at the Mons railroad shops.

Newcastle Virus Conjunctivitis.—Four cases of conjunctivitis following accidental contamination of the conjunctiva in laboratory personnel working with the virus of fowl plague or Newcastle virus are reported (Jacotot, Vallee, and Le Priol [167]; Lepine, Atanasiu, and Gareau [168]). An identical conjunctivitis was

162. Offret, G., and Albahary, C.: Epidemic Keratoconjunctivitis with Occupational Aspect, Arch. mal. profess. **10**:262-263, 1949.

163. Correard, R., and Plessier, P.: Recent Epidemic of Keratoconjunctivitis in the Paris Area, Ann. ocul. **182**:859-863, 1949.

164. Capalbi, S.: Treatment of Epidemic Keratoconjunctivitis, Arch. ottal. **53**:396-401, 1950.

165. Bietti, G.: Results of Treatment with Antibiotics in Virus Diseases of the Eye, Rass. clin.-sc. **25**:73-77, 1949.

166. Hubert and Lebas: Acute Epidemic Follicular Conjunctivitis, Bull. Soc. belge opht. **93**:530-532, 1949.

167. Jacotot, H.; Vallée, A., and Le Priol, A.: A Case of Human Conjunctivitis Due to the Newcastle Virus (Fowl Plague) Contracted in the Laboratory, Bull. Acad. nat. méd. **134**:106-108, 1950.

168. Lepine, P.; Atanasiu, P., and Gareau, G.: Three Laboratory Infections with Newcastle Disease Virus, Ann. Inst. Pasteur **79**:193-196, 1950.

reported by Garcia Miranda [169] in five patients who had handled birds that had died of fowl plague, and by Sheehan [170] in two poultry farmers who had an outbreak of Newcastle disease among their chickens.

The incubation period in the three cases of accidental contamination of the conjunctiva reported by Lepine, Atanasiu, and Gareau was 20 to 30 hours. The ocular lesions were characterized by diffuse subconjunctival hemorrhages, chemosis, preauricular lymphadenopathy preceded by general symptoms, and slight fever. The lesions subsided in six to eight days.

Sheehan reports prompt relief after local use of aureomycin; but Lepine, Atanasiu, and Gareau believe that aureomycin did not alter the course in their cases. They also state that the instillation of 2% mild silver protein N. F. did not serve as a prophylactic.

Herpes Zoster.—Forsius [171] reports two cases of generalized herpes zoster originating from ophthalmic herpes zoster. One patient was a man aged 69, and the other, a man aged 26. He also reports two cases of severe ophthalmic herpes zoster in elderly women, with six and nine blisters, respectively, on the body. This condition is rarely described in the literature.

Ross [172] reports two cases of herpes zoster ophthalmicus without cutaneous eruptions. In the first case, that of a man aged 66, severe neuralgia of the right side of the head and face was followed by preauricular lymphadenopathy, episcleritis, disciform keratitis, and iritis. After a stormy course of two weeks, the third nerve became completely paralyzed. Two weeks later severe glaucoma developed, necessitating enucleation. In the second case, that of a man aged 50, the disease ran a similar course, but glaucoma did not develop. The author thinks that ophthalmic herpes zoster is commoner than the literature would indicate.

Chickenpox.—Frandsen [173] observed two cases of deep keratitis which developed 10 days after the general eruption in chickenpox. In one case the lesion disappeared in two weeks; in the other the condition developed into typical disciform keratitis.

Vaccinia—Dreisler [174] describes four cases of unilateral infection of the eye with smallpox vaccine. Two patients exhibited typical pustules of the lid margin, and another, subepithelial keratitis and moderate iridocyclitis. The fourth patient, a woman aged 66, had been vaccinated as a child, and not again until the virus was transferred to her eye by a newly vaccinated child. In addition to lesions of the skin and conjunctiva, there developed severe iridocyclitis and exophthalmos, result-

169. Garcia Miranda, R.: Newcastle Virus and the Eye: Clinical Studies and Experimental Tests, Arch. Soc. oftal. hispano-am. **9**:720-725, 1949.

170. Sheehan, G. S.: Newcastle Disease May Cause Human Conjunctivitis, J. Am. Vet. M. A. **116**:219, 1950.

171. Forsius, H.: Herpes Zoster Generalisatus with Complications of the Eye, Ophthalmologica **119**:285-291, 1950.

172. Ross, J. V. M.: Herpes Zoster Ophthalmicus Sine Eruptione, Arch. Ophth. **42**:808-812 (Dec.) 1949.

173. Frandsen, E.: Chickenpox in the Cornea, Acta ophth. **28**:113-120, 1950.

174. Dreisler, K. K.: Eye Complications After Smallpox Vaccination, Ugesk. læger **111**:850-852, 1949.

ing in loss of vision and symblepharon. Hartleib [175] reports two cases of vaccinia affecting the lids.

Molluscum Contagiosum.—Van der Meer Maastricht and Gomperts [176] describe a tumor which involved the whole inferior eyelid but was not attached to the tarsus. Histologic examination after excision revealed molluscum contagiosum.

Epidemic Parotitis.—Michalek and Iserle [177] describe a case of keratitis profunda which developed in a boy aged 13 on the fifth day after he noticed swelling of both parotid glands.

Parasites.—Although cysticercosis of the eye most commonly produces a subretinal cyst, Melanowski,[178] during the last 26 years, has observed cases in which the lesion occurred in the eyelids and under the conjunctiva.

Castresana y Guinea [179] observed an increase in frequency of infection with Filaria palpebralis in Spain in the last few years. He warns of the possibility of migration of fresh parasites in patients in whom ocular localization has occurred.

Puig Solanes, Riveroll Noble, and Fonte [180] made examinations in over 1,000 cases of onchocerciasis. In the acute form of the disease, they describe intense irritation of the skin of the eyelids, with swellings, blepharospasm, lacrimation, and injection of the anterior segment. In the chronic form of the disease all tissues of the eye are involved. The conjunctiva shows vascularization and pigmentation. Other lesions include keratitis, iritis with paresis of the pupil and iris atrophy, and choroidal and retinal atrophy. The filarias apparently penetrate the globe from external sources.

Cogan and Grant [181] report the effective use of anticholinesterase agents in killing pediculi on lashes of the eye.

Ocular myiasis may be confined to larval conjunctivitis in the case of Oestrus ovis if the larvae are removed promptly. Lorente Buesa [182] reports the removal of 15 larvae from one patient, and Conway,[183] the removal of 7 to 13 larvae from the conjunctivas of two aircraftsmen and one British soldier in the Middle East air force. The conjunctivitis subsided in one to three days after removal of the larvae in each case.

Friedmann [184] reports the first case of thelaziasis conjunctivae observed by him in South India. The parasite occurred in the left eye of a girl aged 1½ years.

175. Hartleib, R.: Vaccinia Affecting the Eye, Med. Klin. **44**:994-996, 1949.

176. van der Meer Maastricht, B. C. J., and Gomperts, C. E.: Molluscum Contagiosum Giganteum, Am. J. Ophth. **33**:965-967, 1950.

177. Michalek, V., and Iserle, J. Z.: Otitic and Ophthalmic Complications of Epidemic Parotitis, Časop. lék. Česk. **88**:1509-1512, 1949.

178. Melanowski, W. H.: Cysticercosis of the Eye, Tr. Ophth. Soc. U. Kingdom **67**:229-239, 1948.

179. Castresana y Guinea, A.: Filaria Palpebralis, Arch. Soc. oftal. hispano-am. **9**:293-305, 1949.

180. Puig Solanes, M.; Riveroll Noble, B., and Fonte, A.: Ocular Onchocerciasis, Am. J. Ophth. **32**:1207-1212, 1949.

181. Cogan, D. G., and Grant, W. M.: Treatment of Pediculosis Ciliaris with Anticholinesterase Agents: Report of a Case, Arch. Ophth. **41**:627-628 (May) 1949.

182. Lorente Buesa: A Case of Ocular Myiasis by Oestrus Ovis, Arch. Soc. oftal. hispano-am. **10**:145-150, 1950.

183. Conway, J. S.: Larval Conjunctivitis, Brit. M. J. **2**:928, 1950.

184. Friedmann, M.: Thelaziasis of the Conjunctiva, Ophthalmologica **118**:193-204, 1949.

Previously, seven cases had been reported from China, Korea, and the Philippine Islands and two from California. The Thelazia callipaeda is a filaria-like worm living in the conjunctival sac of the horse, ox, buffalo, pig, camel, dog, cat, sheep, black bear, rabbit, wild birds, and man. The worm is whitish, thin, thread-like, and transparent and measures 7 to 20 mm. in length and 0.5 mm. in breadth.

Kumar Ghose [185] found an unusual foreign body in the lower fornix of a patient —a small snail.

Witenberg, Jacoby, and Steckelmacher [186] report the removal of a larva of the nematode Gnathostoma spinigerum from the limbic region of the left eye of a man aged 40.

Vernal Conjunctivitis.—Rossi and Fornaro [187] conclude from their observations on the course of vernal conjunctivitis after the administration of corticotropin that the disease depends upon a complex combined endocrine disorder, with factors of hypoadrenalism, hypogenitalism, and hyperthymism. Lippi [188] believes that these factors are frequently present but that an allergic diathesis is primary.

Edmund [189] reports a case of vernal conjunctivitis combined with Besnier's prurigo and emphasizes the similarity of the lesions.

A special fibropapillary vascular pannus is described by Pascheff [190] in some cases of relapsing vernal conjunctivitis. It develops concentrically like an arcus senilis but does not leave a transparent zone at the limbus. The conjunctiva of the limbus is not hypertrophied, but a pigmented circular zone is formed at the level of the limbus. Histologic examination reveals two kinds of melanocytes: pigmented basal cells and pigmented cells with fine dendritic processes lying between the basal cells. Girones [191] reports a case of this type in a woman aged 22, emphasizing that the condition is a chronic fibropapillary hyperplasia which has a serious invasive character and may end in blindness.

Various methods of treatment of vernal conjunctivitis are reported. Hart and Dimitriou [192] give small doses (100 r) of roentgen radiation to the spleen once a week for four to six weeks. The subjective symptoms become less intense in one week and disappear after the second treatment. The perilimbic manifestations generally disappear, and the palpebral lesions improve. The authors do not believe that the treatment is curative, but the results are effective for one season. Sokolik [193]

185. Kumar Ghose, N.: Unusual Ocular Foreign Body, Brit. J. Ophth. **33**:520-521, 1949.

186. Witenberg, G.; Jacoby, J., and Steckelmacher, S.: A Case of Ocular Gnathostomiasis Ophthalmologica **119**:114-122, 1950.

187. Rossi, A., and Fornaro, L.: Circulating Eosinophils in Vernal Conjunctivitis Under the Action of Corticotropin (ACTH), Arch. ottal. **53**:223-238, 1949.

188. Lippi, E. V.: Patients with Vernal Conjunctivitis, Arch. ottal. **53**:257-288, 1949.

189. Edmund, J.: Vernal Conjunctivitis Combined with Prurigo Besnier: Report of a Case, Acta ophth. **28**:215-222, 1950.

190. Pascheff: Concentric and Progressive Annular Form of Pannus Vernalis, Arch. opht. **9**:596-598, 1949; The Dendritic Melanocyte of the Conjunctiva of the Limbus, ibid. **9**:599, 1949.

191. Girones, E.: Pannus of Vernal Conjunctivitis in the Concentrically Progressive Annular Form, Described by Pascheff, Arch. Soc. oftal. hispano-am. **9**:1228-1230, 1949.

192. Hart, E., and Dimitriou, T.: X-Ray Findings in Vernal Conjunctivitis, Bull. Soc. hellen. ophth. **17**:254-270, 1950.

193. Sokolik, E.: Injections of Cod Liver Oil in the Treatment of Vernal Conjunctivitis, Československ. oftal. **6**:39, 1950.

reports improvement in five cases of vernal conjunctivitis after a series of 5 to 10 intramuscular injections of 1 ml. of cod liver oil prepared by the method of Filatov. On the basis of recent indications that riboflavin has antihistaminic activity, Stern [194] reports the treatment of 22 patients with vernal conjunctivitis by administration of 10 mg. of riboflavin daily by mouth. Patients with milky edema of the conjunctiva only and those with the bulbar and mixed types did not respond, but nine patients with papillary hypertrophy improved uniformly without any other treatment. Thus, the author concludes that other factors are primarily important but that riboflavin is a valuable adjunct in symptomatic treatment of this disease.

Allergy.—In addition to general articles on ocular allergy by Hansen [195] and by Melanowski,[196] Ziegler [197] describes a case of Mikulicz syndrome which he believes to be allergic on the basis of clinical and histologic findings. Costi [198] reports cases of subacute conjunctivitis in which the disease was intractable to local treatment of septic nasal foci. Mata [199] reports two cases of polymicrobic allergy producing epiphora, due to edema and obstruction of the opening of the lacrimal duct, without other signs of nasal allergy. In both cases cure was effected by appropriate vaccine therapy.

Fralick and Kiess [200] reported that administration of 50 mg. of tripelennamine ("pyribenzamine") hydrochloride by mouth four times a day and local applications of 0.25 or 0.5% diphenhydramine (benadryl®) hydrochloride solution four times a day will permit the continued use of atropine in cases in which atropine dermatitis and conjunctivitis have developed; of patients so treated, two-thirds were relieved of symptoms.

Strandberg [201] reports successful treatment of two children with bronchial asthma and conjunctivitis by instillation of 5% ephedrine hydrochloride onto the conjunctiva.

Peiffer [202] used antistin® (2-[N - phenyl - n - benzylaminomethyl] - imidazoline) successfully in a number of cases of allergic disease of the eye. Lohr [203] reports symptomatic improvement after the use of drops of 1/1000 or 1/10,000

194. Stern, H. J.: Riboflavin Treatment of Spring Catarrah, Am. J. Ophth. **32**:1553-1556, 1949.

195. Hansen, E. W.: Allergy in Ophthalmology, Tr. Am. Acad. Ophth. **54**:299-311 (March-April) 1950.

196. Melanowski, W. H.: Allergy in Eye Diseases, Polski tygodnik lek. **5**:1089-1099, 1950.

197. Ziegler, A. I.: Contribution to the Problem of Mikulicz' Syndrome, Wien. klin. Wchnschr. **61**:722-724, 1949.

198. Costi, C.: Allergic Conjunctivitis Due to a Nasal Septic Focus, Arch. Soc. oftal. hispano-am. **9**:1293-1303, 1949.

199. Mata, P.: Epiphora of Bacterial Allergic Origin, Arch. Soc. oftal. hispano-am. **9**:1285-1292, 1949.

200. Fralick, F. B., and Kiess, R. D.: Use of Antihistaminic Drugs in Control of Atropine Dermatitis and Conjunctivitis, Arch. Ophth. **41**:583-586 (May) 1949.

201. Strandberg, B.: Two Cases of Allergic Conjunctivitis Treated Locally with Ephedrine, Ugesk. læger **112**:634, 1950.

202. Peiffer, H.: Treatment of Allergic Diseases of the Eye with Antistin,® Klin. Monatsbl. Augenh. **116**:293-304, 1950.

203. Lohr, K.: Indications and Methods of Naphazoline (Privine®) Therapy of the Eye, Klin. Monatsbl. Augenh. **116**:285-292, 1950.

naphazoline (privine®) hydrochloride solution. Knupffer [204] found that naphazoline or antistin® and naphazoline shortened the conjunctival reaction in cases of artificially produced allergic conjunctivitis, whereas astringent solutions were practically without effect.

Robison [205] suggests that, since corneal sensitivity is less frequent than that of the skin and conjunctiva, contact conjunctivitis may be prevented by applying atropine only to the cornea. He uses a tubular corneal applicator for this purpose, protecting the skin with a heavy coating of white petrolatum U. S. P. and the conjunctiva by the instillation of iodized sesame oil. At the end of the application the surplus atropine is removed from the applicator by the instillation of iodized sesame oil into it, and the surface of the oil is irrigated with saline solution.

Conjunctival Tumor.—Ash [206] analyzed 1,120 specimens of epibulbar tumors and found 346 epithelial tumors, 244 nevi, 60 malignant melanomas, 69 dermoids and teratomas, 27 other benign neoplasms, 89 pterygia and pingueculas, and 78 other lesions. In the 12 cases of lymphoma, the predominant location was in the conjunctival fornix.

Epithelial cysts of the limbus may arise from finger-like epithelial ridges stretching radially from the cornea into the adjacent conjunctiva, according to Kornerup,[207] who reports seven cases of this type.

A large, sausage-like, traumatic subconjunctival implantation cyst, extending from 6 o'clock around to 3 o'clock near the limbus, collapsed and did not reform after Werner [208] excised approximately one-third of it.

Di Prima,[209] in reporting a case of primary spindle-cell epithelioma of the limbus successfully removed from a man aged 58, stresses the importance of conservative surgery.

A papillary epithelioma of the tarsal conjunctiva of a man aged 70 was treated by contact application of 5,000 r of roentgen radiation to the averted lid. Offret [210] observed no recurrence after four years.

Magni [211] describes three cases of reticulohistiocytoma of the conjunctiva, the epibulbar conjunctiva being involved in two cases and the lower fornix in one. Brognoli [212] reports a conjunctival endothelioma occurring near the limbus in a boy aged 4½ years.

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204. Knupffer, N.: Results of Treatment of Allergic Conjunctivitis with Naphazoline (Privine®) and Antistin®-Naphazoline, Klin. Monatsbl. Augenh. **116**:304-309, 1950.

205. Robison, J. M.: A Method of Preventing Contact Conjunctivitis, Am. J. Ophth. **33**:964-965, 1950.

206. Ash, J. E.: Epibulbar Tumors, Am. J. Ophth. **33**:1203-1219, 1950.

207. Kornerup, T.: Epithelial Cysts at the Limbus Conjunctivae in Man, Acta ophth. **27**:483-506, 1949.

208. Werner, S.: A Peculiar Case of Subconjunctival Implantation Cysts on the Globe, Acta ophth. **28**:203-208, 1950.

209. di Prima, J.: A Case of Primary Spindle-Cell Epithelioma of the Sclerocorneal Limbus with Rapid Evolution (Histologic Notes), Rass. ital. ottal. **19**:348-356, 1950.

210. Offret, G.: Successful Treatment of a Papillary Epithelioma of the Tarsal Conjunctiva by Contact Roentgen Irradiation, Bull. Soc. opht. Paris **4**:157-159, 1950.

211. Magni, S.: Reticulohistiocytoma of the Conjunctiva, Gior. ital. oftal. **2**:427-445, 1949.

212. Brognoli, C.: Contribution to Knowledge of Conjunctival Endotheliomas, Boll. ocul. **28**:349-360, 1949.

Pingueculas and pterygia usually contain lipoid-bearing structures which fluoresce in ultraviolet radiation. Wilson [213] uses the Hildreth lamp to help differentiate these lesions from lipoid-free malignant growths of the conjunctiva. However, he warns that some malignant lesions may show fluorescence and some nonmalignant lesions may fail to show this phenomenon, but he advises that all nonfluorescent lesions should be examined histologically.

An unusual case of Bowen's disease was reported by Mackie and Edwards.[214] The lesion developed in a conjunctival flap covering a Mooren ulcer (rodent ulcer of the cornea). François, Kluyskens, and Rabaey [215] used contact radium therapy in another case. This is the second case on record in which the lesion has responded to this form of treatment.

According to Toulant and Foissin,[216] conjunctival plasmocytoma is a yellowish-red, unilateral solitary tumor localized on the palpebral conjunctiva of the upper lid. Trachoma coexisted in 80% of their 25 cases. The lesions were treated by simple excision.

Wolff [217] challenges Masson's theory of the neuroectodermal origin of nevi because epithelial downgrowths in these tumors may proliferate and perhaps produce malignant growths and because he is of the opinion that the tactile cells of the Merkel-Ranvier corpuscles are derived from epithelium.

A malignant melanoma arising in a dermal-epidermal nevus of the bulbar conjunctiva of the right eye of a boy, aged 10 years, is reported by Kesinger.[218] A similar nevus was removed from the left eye and found to be potentially malignant. Both lesions were noticed when the boy was 14 months of age, but the one in the right eye had begun to enlarge a few months before it was removed.

Puchner [219] excised a melanocarcinoma of the caruncle of a man aged 51, and Galindez Iglesias [220] described a case of bilateral melanosis of the conjunctiva.

Plastic Surgery.—Majoros [221] cuts a conjunctival graft 5 mm. square and places it at the site of a thoroughly scraped and cleansed pterygium wound. He does not use sutures but has the patient keep his eyes closed for 10 to 15 minutes after the

213. Wilson, C.: Differentiation of Conjunctival Tumors, Am. J. Ophth. **32**:1407, 1949.

214. Mackie, E. G., and Edwards, J. L.: A Case of Bowen's Disease of the Eye, Tr. Ophth. Soc. U. Kingdom **68**:539-544, 1949.

215. François, J.; Kluyskens, J., and Rabaey, M.: Intra-Epithelioma of the Conjunctiva and the Cornea (Bowen's Disease) Healed by Contact Radiotherapy, Brit. J. Ophth. **34**:360-364, 1950.

216. Toulant, P., and Foissin, J.: Conjunctival Plasmocytoma, Bull. Soc. opht. France **62**:157-160, 1949.

217. Wolff, E.: The Conjunctival Naevus and the Neurogenic Theory of Melanomata, Tr. Ophth. Soc. U. Kingdom **68**:213-223, 1949.

218. Kesinger, H. F.: Malignant Melanoma Arising in a Dermal Epidermal Nevus of the Bulbar Conjunctiva: A Case Report, Am. J. Ophth. **33**:967-969, 1950.

219. Puchner, A. F.: Melanocarcinoma of the Lacrimal Caruncle, Vestnik oftal. **28**:41-42, 1949.

220. Galindez Iglesias, F.: Melanosis of the Eyeball, Predominantly in the Conjunctiva, Bol. Inst. pat. méd. **4**:108-113, 1949.

221. Majoros, J.: Conjunctival Transplant for Treatment of Pterygium, Am. J. Ophth. **33**:1289, 1950.

graft is positioned. In 2 out of more than 50 operations the transplant became necrotic and caused a severe reaction, but 50 transplants were made by this technique without a recurrence.

Several tissues have been used successfully to replace conjunctiva. Labial mucosa and rabbit conjunctiva were used by Rebello [222]; amniotic membrane, by Dansey-Browning,[223] and free epidermal grafts over a stent, by Grdovic.[224] Cima [225] used cadaver conjunctiva but had two cases in which the transplants were not tolerated. The tissue was expelled from the site of the graft in a short time.

Wong and Slaughter [226] report subconjunctival hemorrhages in 32 of 55 rhinoplastic surgical procedures. The hemorrhages appeared within 24 to 36 hours after the operation and were located only in the temporal sectors of the bulbar conjunctiva in 31 cases.

222. Rebello, O.: Conjunctival Grafts, Rev. brasil. cir. **18**:869-874, 1949.

223. Dansey-Browning, C.: Use of Amniotic Membrane, Brit. J. Ophth. **33**:518-520, 1949.

224. Grdovic, M.: Plastic Operation on the Conjunctival Sac, Liječn. vjes. **71**:194-196, 1949.

225. Cima, V.: Interpretation of Some Peculiar Reactions to Grafts of Conjunctiva from Cadavers, Boll. ocul. **28**:155-160, 1949.

226. Wong, W. W., and Slaughter, W. B.: Temporal Subconjunctival Hemorrhages as a Complication of Rhinoplastic Procedures, Am. J. Ophth. **32**:973-981, 1949.

Society Transactions

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

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M. Luther Kauffman, M.D., *Clerk*

Regular Meeting, March 15, 1951

000000 Chromic Absorbable Surgical Suture U. S. P. (Surgical Gut) in Wound Closure After Cataract Extraction. Dr. F. Bruce Fralick, Ann Arbor, Mich.

Dr. Fralick gave an account of his experimental work and experience with absorbable surgical suture U. S. P. (surgical gut U. S. P. XIII) for general use in ocular surgery. The complete text of his paper can be found in the *West Virginia Medical Journal* of September, 1950, page 251.

DISCUSSION

Dr. Edmund B. Spaeth: By mild chromic surgical gut, does Dr. Fralick mean the 10-day or the 20-day kind? Are these of approximate duration?

Dr. Bruce Fralick, Ann Arbor, Mich.: Mild chromic surgical gut is of approximately 10 days' duration.

Dr. Edmund B. Spaeth: Would Dr. Fralick tell us how he handles his complications in cataract surgery, so far as suturing is concerned, and perhaps discuss how he uses sutures for corneal lacerations not necessarily connected with cataract surgery?

Dr. Bruce Fralick, Ann Arbor, Mich.: My colleagues and I use 000000 mild chromic surgical gut in all cases of cataract surgery, both for the corneoscleral wound and the conjunctival flap. With loss of vitreous, it is necessary when using absorbable surgical sutures to make a surgeon's knot, since the suture becomes so slippery when covered with vitreous that the ordinary square knot will not hold. The surgeons' knot is somewhat larger, but we have not noted any unusual irritation from its presence.

We used 000000 mild chromic surgical gut to close corneal lacerations where direct suturing was thought advisable. Likewise, ruptures of the sclera are closed with buried surgical gut. We much prefer surgical gut sutures to silk sutures in repairing lacerations of the lid, closing layer by layer with surgical gut, but closing the skin wound with silk.

Dr. I. S. Tassman: I should like to ask a question, but before I do, I wonder if I may be permitted a comment on Dr. Fralick's reference to the delayed proliferation of fibroblasts which occurs, sometimes after four days. I have sections which show that the use of plasma and thrombin after cataract section produces proliferation of fibroblasts within 24 hours, as compared with the delayed proliferation in control sections of eyes in which the plasma and thrombin were not used. I do not know what the effect of plasma and thrombin may be on surgical gut sutures, but I do know that it reinforces, and gives more immediate sealing of the wound in association with, silk sutures.

As Dr. Fralick stated, the silk sutures may not hold tight as long as six days, but I am sure that the wound will be closed in 24 hours with the use of plasma and thrombin solutions.

My question is this: In such cases as those of a dislocated lens or increased pressure on the eye, what security do you have when you have reason to expect an immediate expulsion of lens or an escape of vitreous when the section is made? How much security do you get, and what benefit is obtained during the course of the operation, from all the postplaced sutures? For postoperative security, closure of the wound with a silk suture fortified by the use of plasma and thrombin provides the desired closure and much earlier wound healing.

DR. BRUCE FRALICK, Ann Arbor Mich.: Dr. Tassman has made a good criticism regarding the worthlessness of postplaced sutures as a safeguard against expulsion of the lens or vitreous during the making of the section. The cornea and sclera are tough, and it is not easy to pass the best needles obtainable through this tissue. When vitreous is lost with the section, the placing of the sutures at the time is likely to lead to further loss of vitreous. The same criticism applies to silk as to surgical gut when postplaced sutures are used. Quite likely, the preplaced suture, such as the McLean or the Verhoeff type, would allow immediate closure of the wound and prevent further loss of vitreous.

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Stanford University School of Medicine Postgraduate Conference in Clinical Ophthalmology.—The annual Postgraduate Conference in Clinical Ophthalmology will be presented by the Stanford University School of Medicine from March 24 through March 28, 1952. The program this year will be devoted to ophthalmic surgery. Registration will be open to physicians who limit their practice to the treatment of diseases of the eye or the eye, ear, nose, and throat. In order to allow free discussion by members of the conference, registration will be limited to 30 physicians.

Instructors will be Dr. A. Edward Maumenee, Dr. Dohrmann K. Pischel, Dr. Jerome W. Bettman, Dr. Max Fine, Dr. Earle H. McBain, and Dr. Arthur J. Jampolsky.

Programs and further information may be obtained from the Office of the Dean, Stanford University School of Medicine, 2398 Sacramento St., San Francisco 15.

The Retina Foundation.—The Retina Foundation, founded in April, 1950, has now been organized as a new basic research unit of the Massachusetts Eye and Ear Infirmary. The Foundation, located at 30 Chambers St., Boston, includes laboratories for biochemistry, electron microscopy, tissue culture, and optics. The members of the staff are Charles L. Schepens, M.D., director, an ophthalmologist interested in clinical work in retinal detachment; E. A. Balazs, M.D., associate director, a histochemist interested in the study of mucopolysaccharides and tissue growth; Marie A. Jakus, Ph.D., a biologist interested in the study of fine structure, and F. Wachendorf, D. Eng., a physicist specializing in optics.

The members of the research advisory committee of the Foundation are T. Gundersen, M.D., chairman; P. Boeder, Ph.D.; P. A. Chandler, M.D.; W. J. Crozier, Ph.D.; P. Heath, M.D., and B. Sachs, M.D.

The Retina Foundation has received grants from the United States Public Health Service, the American Optical Company, the Kresge Foundation, the G. M. Hyams Fund, Inc., the Vivian B. Allen Foundation, the National Council to Combat Blindness, Inc., the Office of Naval Research, and the Committee of the Permanent Charity Fund, Inc. It is also supported by donations from interested persons. In addition, the work was supported in its initial stages by the Veterans Administration.

University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology.—The sixth annual University of Florida Midwinter Seminar in Ophthalmology and Otolaryngology will be held in January, 1952, convening Jan. 14 and continuing through Jan. 19. As in previous years, Miami Beach will again be the host city.

The lectures on ophthalmology will be presented Jan. 14, 15, and 16; those on otolaryngology, on Jan. 17, 18, and 19. Lecturers for the courses in ophthalmology will include such distinguished medical teachers as Dr. Alson E. Braley, Iowa City; Dr. John M. McLean, New York; Dr. Wendell Hughes, Hempstead, New York; Dr. Irving H. Leopold, Philadelphia, and Dr. Bruce Fralick, Ann Arbor, Mich.

Outstanding otolaryngologists who will lecture include Dr. Fred W. Dixon, Cleveland; Dr. Thomas D. Galloway, Evanston, Ill.; Dr. Anderson G. Hilding, Duluth, Minn.; Dr. Moses H. Lurie, Boston, and Dr. Henry L. Williams, Rochester, Minn.

On Wednesday evening, Jan. 16, all registrants are cordially invited to attend the Midwinter Convention of the Florida Society of Ophthalmology and Otolaryngology, which meets concurrently with the Seminar. A certificate of attendance at the Convention will be issued for deduction purposes. Outstanding discussions will be presented at this time.

All meetings will be held at the Sans Souci Hotel, Miami Beach, Fla. Dr. Walter T. Hotchkiss, Miami Beach, Fla., is secretary, and communications concerning the seminar should be addressed to him.

Obituaries

WALTER BRACKETT LANCASTER, M.D.
1863-1951

Out of the midst of an active existence, which he carried on to the very end of his days in spite of his advanced age, Walter B. Lancaster was taken away by a merciful death that came to him unannounced during the night of Dec. 8, 1951. With his death a major figure of American ophthalmology has passed into history. Such was his stature that it can truly be said that everyone specializing in ophthalmology in America during the past 30 years has felt his influence directly or indirectly.

Lancaster was born in Newton, Mass., on May 11, 1863, into a prosperous family of old New England stock. After attending the local schools, he entered Harvard University, graduating magna cum laude in 1884, and then entered Harvard Medical School. Because of poor health, he was out two years and so did not graduate until 1889. Meanwhile, in December, 1885, he had married Emma Winter, by whom he had a daughter, Julia E. Lancaster, now orthoptist in San Francisco.

After graduation, Lancaster went to Europe, where he studied in Vienna under Mauthner. The inspiration which he received from this master in the fields of refraction and ocular motility was one of the major influences in Lancaster's life. From Vienna, Lancaster went to Paris, Edinburgh, and London and then returned to the United States.

He began general practice in Wellesley, Mass., and also opened an office for the special practice of ophthalmology in Boston. After five years he gave up general practice to devote himself fully to ophthalmology.

In 1901 Lancaster's health, always delicate, became worse and he had to retire from practice. He moved to Amherst, Mass., remaining there until 1910, when he went to England. There he had his appendix removed, and this operation was the turning point in Lancaster's life. His health improved, and, although he remained under the shadow of somewhat precarious health, which placed certain restrictions on him, his activity was never again interrupted; in fact, it became ever increasing. Thus, Lancaster's most active life began when he was well along in middle age. He accomplished between the years of 60 and 85 the work which most men expect to do in the years between 35 and 60.

Upon returning home from England, Lancaster reopened his Boston office, not to give it up again except for the time of his service in the first world war and during the years (1940-1942) which he spent in Hanover, N. H., as chief of staff of the Dartmouth Eye Institute.

During World War I he was a major in the Medical Corps, serving as Chief of the Department of Head Surgery at Camp Devens and Assistant Commanding Officer of the Base Hospital, and later as Head of the Department of Ophthalmology and Assistant Commanding Officer of the Research Laboratory of the Air Force Service. It was while on duty in this laboratory that he interested Dr. Howard in the matter of measurement of stereopsis, which led to the development of the well-known Howard-Dolman device.

Lancaster had always shown keen interest in the work of Adelbert Ames Jr., who had founded a department for research in physiologic optics at Dartmouth College, and he assisted Ames for years with his advice. He was also instrumental in bringing Bielschowsky to Hanover, N. H. Lancaster was, therefore, the logical choice to succeed Bielschowsky after the latter's death. Unfortunately, Lancaster's plans for the development and running of the Dartmouth Eye Institute did not agree with those of its board of directors, and he returned to his Boston practice after a brief stay in Hanover.

This, in outline, is the history of Lancaster's life. The abundance which filled this simple framework is so great that it can only be sketched here.

WALTER BRACKETT LANCASTER, M.D.
1863-1951

His first and uppermost interest was his chosen specialty of ophthalmology. Although he grew up at a time when most of the great minds in ophthalmology concentrated their efforts on the study of anatomy and pathology, Lancaster from the first recognized the importance of a physiologic way of thinking. His personal contributions related predominantly to problems of accommodation, refraction, and physiologic optics, and, more especially, to motor neuro-ophthalmology, the mechanistic approach to which he abhorred; but he always maintained a lively interest in other fields of ocular physiology and kept amazingly well informed about their progress. Yet this interest was never theoretical. Eminently practical and primarily a clinician, Lancaster never studied ocular diseases or anomalies for their own sake, as so many of us do. He preferred to direct his efforts toward affections which permitted a therapeutic approach. This is probably one of the reasons that he always maintained a lively interest in operative technique. A skilful and meticu-

lous surgeon, his thoughts were constantly occupied with ideas for new procedures and the improvement of old ones. At times these ideas were quite daring; only few of them got into print.

To Lancaster's lifelong interest in the teaching of ophthalmology and in the training of young ophthalmologists is due one of his lasting contributions. Even before retiring to Amherst, he had offered courses in clinical ophthalmology, refraction, and ophthalmic surgery at the Boston Polyclinic, and after his return from England he gave for a while at Harvard Medical School a graduate course in physiologic optics, a subject which he considered much neglected.

When Dr. Edward Jackson conceived the idea of a board for examinations in ophthalmology, Lancaster participated in the creation of the American Board of Ophthalmology, in 1915, which set the pattern for all the specialty boards to follow. Lancaster served on it in various capacities until his death, having repeatedly been its president, and he was influential in formulating its policies. There is no doubt that the Board has contributed more than any other single factor in bringing to the attention of the profession the inadequacies in the preparation of young ophthalmologists and the need for their correction. Many outstanding ophthalmologists have unselfishly and unstintingly given their time to the Board, but no one more persistently and actively than Walter B. Lancaster.

For lack of support from the organizations with which he was connected, Lancaster did not have the chance to teach as much as he would have liked. He saw the opportunity to have his wish fulfilled when, after the second world war, the demand for basic training in ophthalmology by far exceeded the number of courses then available. It was an amazing spectacle to watch him, at the age of 83, throw himself with youthful ardor into the task of organizing singlehanded the Ophthalmological Study Council, which would offer such a course, set up according to his conceptions, and which is now known as the Lancaster Course. The hundreds of participants who took this course can testify as to how noticeable Lancaster's influence was in every detail of the organization. This course also afforded him the opportunity of fulfilling another dream which he had had for years, namely, to write a book on optics for ophthalmologists which would impart the necessary knowledge of this subject to the young ophthalmologists in the form which he considered most effective. It is regrettable that he did not see this book in print; it is to appear within a few weeks.

Not content with creating the course for ophthalmologists, Lancaster proceeded in 1948 to organize a course for orthoptists, in cooperation with the American Orthoptic Council. This course, too, has become a permanent institution. It has done much good in the past and will without doubt continue to do so.

The organization of the course for orthoptists is a reflection of Lancaster's lifelong attention to problems of ocular motility. For years his name has been associated with this field and he has been looked up to as an authority; he has contributed a very large share to the development of orthoptics in America.

Ophthalmology was more than a profession for Lancaster. It was a cause to which he was devoted. This may explain his great interest in the organizational aspects of ophthalmology and his willingness to work assiduously in all organizations and on many committees. It provided him with a platform to propound and further his ideals. Thus, he was an essential factor and guiding spirit in the American Committee on Optics and Visual Physiology and in the American Orthop-

tic Council; as a member of the Illuminating Engineering Society, he belonged to its Committee on Research; as member of the National Conference on Nomenclature of Disease, he expended a great deal of time and effort contributing to the classification of eye diseases; he was a member of the executive committee of the Advisory Board of Medical Specialties, a member of the editorial board of the *Journal of the Optical Society of America* and a member of the council of the Association for Research in Ophthalmology.

Lancaster acquitted himself of these and of many other assignments in masterly fashion, with enthusiasm and with an unending capacity for work. As a result, many recognitions came to him. He was president of the New England Ophthalmological Society, of the American Ophthalmological Society, and of the American Academy of Ophthalmology and Otolaryngology and chairman of the Section on Ophthalmology of the American Medical Association. In 1939 Dartmouth College conferred upon him the honorary degree of Doctor of Science. On the occasion of his 80th birthday the May 1943 issue of the *American Journal of Ophthalmology*, published as the Lancaster Testimonial Issue, was made up of contributions of his friends and admirers. In 1945 the American Ophthalmological Society bestowed upon him the highest honor at its disposal, the Howe medal.

This survey of Lancaster's professional career would not be complete without mention of his deep interest in the social side of ophthalmology. This was early evident when he was instrumental in founding the Boston Nursery for Blind Babies, of which he was the first consultant. He did his share in the treatment of indigents as ophthalmic surgeon at the Massachusetts Eye and Ear Infirmary and the Boston City Hospital, and he had a great attachment for the National Society for the Prevention of Blindness, of which he was a director and which awarded to him in 1943 the Leslie Dana Gold Medal for Outstanding Service in the prevention of blindness and conservation of vision. His interest in public welfare also was undoubtedly responsible for the close attention which Lancaster paid to the relationship between ophthalmologists and optometrists.

Behind all these professional activities loomed Lancaster's powerful personality. A master of the spoken and written word, with broad knowledge in all fields of human endeavor, a gentleman in the full meaning of the term, he combined science with wisdom, self-assurance with humility. Ever ready with help and advice, he was always willing to learn from anyone: no one was too young; no one's position too insignificant. A strict disciplinarian with himself, Lancaster expected much of others. When principles were at stake, he could be intransigent, and even trenchant. Yet his deep and warm humanity, often expressed in his inimitable humor, was always felt by those close to him.

His death leaves a great void. He will be much missed by his patients, his friends, his colleagues. But the fruits of his work will keep his memory alive. He might well have said, quoting his favorite poet, Horace, which he did so often and so well: *multaque pars mei vitabit libitinam.*

HERMANN M. BURIAN, M.D.

Book Reviews

Perception of the Visual World. By James J. Gibson. Price, $4. Pp. 2,350. Houghton Mifflin Company, 2 Park St., Boston 7, 1951.

This is an interesting book which attempts to answer the question how we see. The physiology of vision can describe the nature of the physical stimulus, the photochemical changes which take place in the retina, and the type of nerve message which the retina signals to the visual cortex. From this point the description of the perceptual processes which explain why things in the world look to us as they do belongs to the field of psychology.

The author of this book is a psychologist, and his approach to the subject is psychological. He is interested primarily in the problems of perception, and not of sensation, and sets out to answer the question of how we get the experience of a concrete visual world from the type of image of the visual field which is presented to the retina.

He proposes a new theory, or hypothesis, which he states "might be called a ground theory, to distinguish it from the air theory which seemed to underlie early research." His basic idea is that "visual space should be conceived, not as an object, or an array of objects in air, but as a continuous surface or an array of adjoining surfaces." This conception is somewhat different from the old belief in the various cues by which we judge depth and distance, both monocularly and binocularly. His theory may be summarized by assuming that the fundamental conditions for seeing a visual world is an array of physical surfaces, reflecting light and projecting on the retina. These surfaces are of two extreme types, frontal and longitudinal. The perception of depth, distance, or the so-called third dimension, is reducible to the problem of perception of longitudinal surfaces. When no surface is present in perception because of homogeneous retinal stimulation, distance is indeterminate. Surfaces are perceived largely on the basis of their texture. The perception of an edge, and hence the perception of a bounded surface in the visual field, is a type of ordinal stimulation (ordinal refers simply to order or succession). This is what has usually, but inaccurately, been called pattern stimulation (consisting of an abrupt transition). The perception of an object in depth is reducible to the problem of the changing slant of a curved surface or the different slant of a bent surface. In either case, the problem is similar to that of how we see a longitudinal surface. The general condition for the perception of a longitudinal or slanted surface is a kind of ordinal stimulation, called a gradient. The author describes the gradients of texture and suggests that other gradients, such as those which are dependent on outlines, or retinal disparity, on shading, on deformation when the observer moves, and possibly on other factors, all have the function of stimulus correlates and yield the impression of distance on a surface. The author builds up each of these factors in some detail and concludes that all the characteristics of visual perception can be derived from the visual stimulus. As he states, "the seeming poverty of the visual stimulus as compared with the richness of visual experience, has apparently been exaggerated." When this stimulus is suitably analyzed, he believes that it is quite adequate to account for the depth and distance of the visual world without the necessity of supposing some special mental process to supplement the images.

To the reviewer, who is not a psychologist, this sounds like a near relative of the gestalt theory. The author develops this theory for the perceptual processes, both when objects and the eye are stationary and during movements of the observer. The latter part of the book is particularly valuable for those interested in aviation medicine. It is, perhaps, unfortunate that the teaching of ophthalmology, even in our best graduate courses, pays so little attention to the psychological aspects of vision. This book should help to remedy that deficiency, and it can be recommended to all ophthalmologists who care to broaden their intellectual horizons.

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Dr. Hermann M. Burian, Iowa City, Iowa; Dr. Alston Callahan, Birmingham, Alabama; Dr. John W. Henderson, Rochester, Minnesota; and, Dr. A. Edward Maumenee, San Francisco, California.

RESIDENT LECTURERS

Dr. F. Bruce Fralick, Dr. Jerome W. Conn, Dr. Harold F. Falls, and Dr. John Woodworth Henderson.

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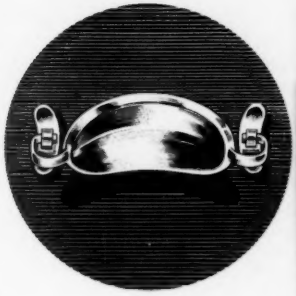

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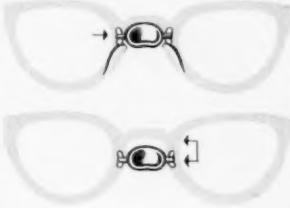

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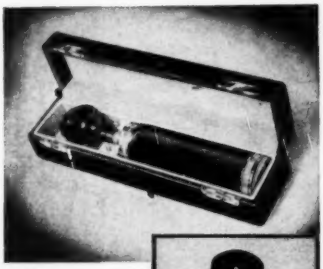

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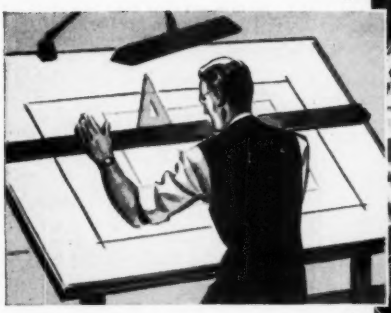

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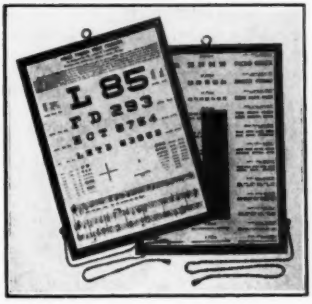

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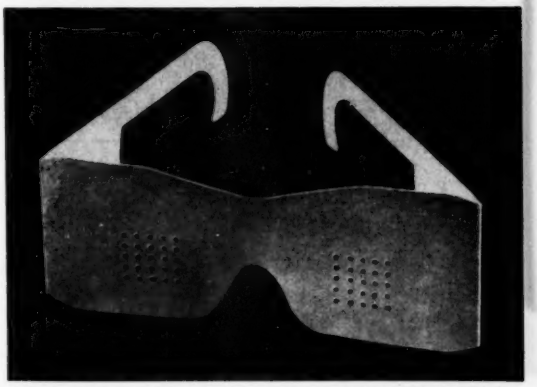

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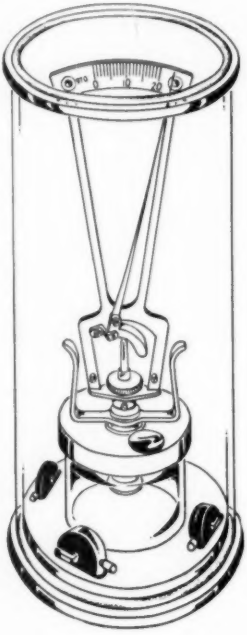

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